THE BRITISH VETERINARY JOURNAL

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Vol. 111 **September, 1955** **No. 9**

EDITORIALS

BELFAST

*PRO TANTO QUID RETRIBUAMUS**

BEAL feirste, meaning the ford at the sand bank, was the original name of the place that has become the great city of Belfast. In ancient times the ford gave a relatively free passage over the tidal river Lagan before it pours out into the great Lough which now bears Belfast's name. Even in medieval times the site was looked upon as only a fortified outpost of Carrickfergus which stands on the northern shore of the Lough. It was during the 17th century that the seeds of this great modern industrial centre were sown by the doughty and industrious colonists from the United Kingdom. They activated and stimulated trade and industry and developed the rich lands of the Valley of the Lagan and gave the town and countryside that robust and loyal character which is exemplified so well to-day.

Hugenot refugees found sanctuary there in the 18th century and taking advantage of the climate and soft streams of water established the yarn and linen industry whose products are famous throughout the world. Taking advantage of the advent of the spinning machine and the decline in raw cotton exports consequent upon the American Civil War, the flax industry was established. Flax, linen and the development of agriculture made possible Belfast, as we know it to-day. Increased trade demanded greater harbour facilities, and shipyards in their turn, and to-day Belfast-built ships are renowned all over the world.

Belfast is a great commercial and industrial city set amidst hills and at the head of a rich valley whose soft winds waft smoke and fog to the sea and keep

*What shall we render in return for so much?

(*The August issue of The British Veterinary Journal was published on August* 8)

it clean and sweet. It is the capital and seat of government of Northern Ireland, a city of fine buildings and a seat of learning with many schools and colleges and a famous University. Those who attended the Congress in 1935 will remember the wonderful hospitality and kindness they received on that occasion, they will recall the joyous and exuberant social events for which our Irish friends are justly famous and the trips to the places of beauty and interest in the glorious counties of Antrim and Down. A congress is justified primarily because of its academic fame. A perusal of the published programme will show that in scope, variety and topicality, the 1955 Congress will not fail through any fault of the local organisers.

AND EVERY DOG HIS DAY

In the programme of the 1955 B.V.A. Congress at Belfast one fails to find the words Dog or Cat. This is remarkable, for there can be no doubt that most Veterinary Surgeons devote much time and effort to the care and treatment of these animals, in sickness and in health, and benefit accordingly in financial return. The omission of specific mention of these two names may be a sign of the times in which we live. Since 1939/40 in consequence of the exigencies of war, government policy and high economic values, the food-producing animals have attained a status and importance that has not been equalled within living memory. Farmers and stock breeders have prospered beyond their wildest dreams and hopes, and as they did in the period of the First World War and during the Napoleonic Wars. One farmer of the latter period when he drove home " well in his cups " after his weekly visit to the market used to sing a song of his own design and whose short chorus was : " Keep on fighting them, Boney ! "

Agricultural prosperity has in turn made its mark on veterinary practice. There has been a great upsurge in the amount of work available. Many " one-man practices " in which before the war a surgeon had a struggle to make a living have benefited to such an extent that several men are able to find plenty of work to do and secure an adequate financial return by devoting their attention to the animals of the farm. The present phase of full employment in the profession is due directly to this change of outlook and opportunity. This is no reason why small animal practice should suffer neglect. Between the wars small animal practice was the salvation of many veterinary surgeons. In many districts the returns from large animal work were insufficient to maintain a professional man. The income derived from the smaller animals acted as a subsidy which enabled him to live in a town or village in reasonable comfort and so ensure that his services could be available for such large animal work as the owners might desire.

There is nothing ignoble in small animal work and there is a great public demand for veterinary service. The work can be of economic and psychological value. The art and science of medicine and surgery can be performed with great precision on the domestic pets, the financial returns are good and, who knows, the time may come again when " we have to go to the dogs," so let us not forget them in this time of farm animal prosperity.

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BRITISH VETERINARY ASSOCIATION

BELFAST CONFERENCE, 1955

SUMMARY OF PROGRAMME

QUEEN'S University, Belfast, is the rendezvous of this year's British Veterinary Association Congress and throughout the week commencing September 4 its halls and lecture rooms will resound to animated lectures and discussions on veterinary medicine.

Visitors to the Congress will be welcomed at the Grand Central Hotel on Sunday, September 4, at an " At Home " given by the North of Ireland Division, and the Lord Mayor of Belfast, Alderman R. J. R. Harcourt, J.P., and Pro-Vice-Chancellor of Queen's, Professor F. H. Newark, B.C.L., M.A., will complete this welcome with speeches on Monday at 10 a.m. at the University. The Minister of Agriculture for Northern Ireland will then formally open the Congress, and Professor A. Robertson, M.A., B.Sc., Ph.D., M.R.C.V.S., F.R.S.E., F.R.I.C., President of the Association, will open the Exhibition of Surgical Instruments, Drugs, Books, etc., in the Great Hall.

One paper will be given on Monday in the afternoon at 2.30 on " Brucellosis Control and Eradication " by Director Woldike Nielsen of Denmark, Dr. C. Mitchell, of Canada, and Dr. C. K. Mingle, of the United States, who will speak on the measures in force in their respective countries.

It is difficult to sum up the programme which has been announced for this 73rd annual congress as it is varied and covers many aspects of veterinary science and practice, but perhaps an interesting feature is the time that has been allotted to papers on the effects of physical conditions and environment on the animal kingdom. This trend in veterinary literature has been in evidence in recent years. Such papers are scheduled as follows:

Wednesday, September 7, 9.30 a.m.: " Impact of Environment on the Health and Productivity of Pigs " by W. A. M. Gordon, M.R.C.V.S., and D. Luke, Ph.D., B.Sc., M.R.C.V.S.

Thursday, September 8, 9.30 a.m.: " The Effect of Atomic Explosions upon Animals " by Dr. F. Louitt (Harwell) and Dr. Scott Russell (Oxford University).

Friday, September 9, 9.30 a.m.: "Adverse Effects on Animal Health brought about by Man's Interference with Nature " by E. L. Taylor, C.B.E., D.V.Sc., M.R.C.V.S., D.V.H., N.D.A., N.D.D. 11 a.m.: " Transport of Animals and Problems Arising " by Major R. C. G. Hancock, B.Sc., M.R.C.V.S., and James Reid, M.R.C.V.S., D.V.S.M.

Statistical methods find increasing favour with veterinary research workers. There can be no doubt that when the known facts and figures appertaining to any problem are subjected to a critical statistical analysis, new light is thrown upon the natural history of disease. On Tuesday, at 9.30 a.m., a symposium of short papers will be given as follows: "Vital Statistics in Veterinary Medicine," "Survey of Diseases of Dairy Cattle," by F. W. Withers, M.A., Ph.D., M.R.C.V.S.; "A Statistical Survey of Veterinary Practice" by C. S. Grunsell, Ph.D., M.R.C.V.S., and H. Paver, Ph.D., B.Sc.; "Survey of Sheep Diseases" by G. B. S. Heath, B.Sc., F.R.C.V.S.; "Survey of Pig Losses" by J. F. Gracey, M.R.C.V.S., D.V.S.M. At certain times two meetings will take place concurrently in different halls and while the above symposium is in progress, J. S. M. Cosgrove, M.R.C.V.S., will present a paper entitled "The Veterinary Surgeon and the New-born Foal."

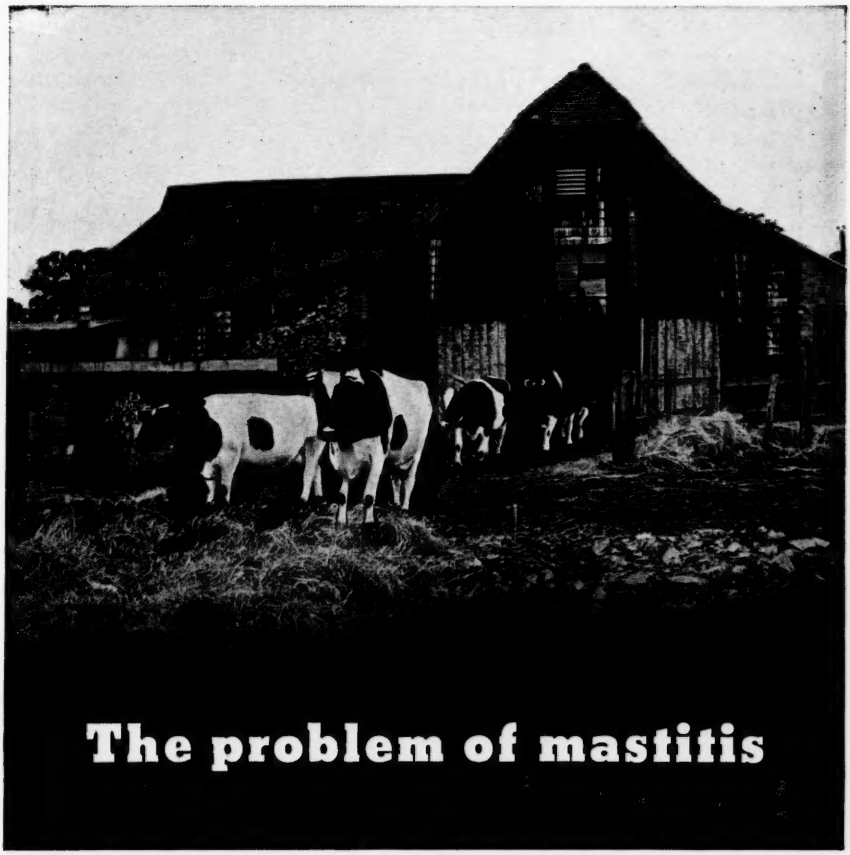
There are three papers dealing with recent advances in veterinary knowledge and these will be given as follows:

Thursday, September 8, 11 a.m.: "Recent Advances in Parasiticides" by H. E. Harbour, B.A., M.R.C.V.S.

Friday, September 9, 11 a.m.: "Recent Advances in our Knowledge of Ruminant Digestion" by A. T. Phillipson, M.A., Ph.D., M.R.C.V.S., F.R.S.E. 2.30 p.m.: "Recent Advances in Poultry Therapeutics" by W. P. Blount, Ph.D., F.R.C.V.S., F.R.S.E.

Also at 2.30 p.m. on Friday what promises to be an interesting paper will be given by Professor E. C. Amoroso, Ph.D., B.Sc., M.B., B.Ch., B.A.O., entitled "Hormone Control of the Œstrous Cycle," and to conclude this summary of the programme mention must be made of one remaining paper on "Safe Anæsthesia" by L. W. Hall, M.A., B.Sc., M.R.C.V.S., on Thursday, September 8, at 11 a.m.

Films will be shown on most days. The programme of social events is an intriguing one and it may well be that the Mountains of Mourne, the Giant's Causeway and Carnlough may tempt even the studious ones to taste other than academic fare. Dances are arranged for each evening with "Ulster Night" on Thursday and the President's Reception and Dance on Friday.



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GENERAL ARTICLES

SOME COMMON FACTORS IN THE PATHOLOGY OF THE NEW-BORN PIG

By R. F. W. GOODWIN, M.A., Ph.D., B.Sc., M.R.C.V.S.
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Introduction

THE neonatal period is a time of special hazard in mammalian development and is associated with heavy mortality in both man and domestic animals. It is difficult, however, to compare the neonatal mortality rates in different species when the environmental and biological influences vary so greatly between them.

An example of the rôle of environment is provided by the various mortality rates for human infants, for in those countries such as Britain, where infant losses have long attracted professional and public attention, the last half-century has witnessed a great reduction in the mortality rate for infants under one year, together with a substantial reduction in the neonatal mortality rate: yet among the primitive communities of the world the problem of neonatal and infant mortality is still comparable with that in animals. In recognising that this problem has responded to environmental changes in man, it is instructive to note that the improvement from the high mortality rates of the last century has derived mainly from reducing the mortality among the older infants. Much less impression has been made on the mortality occurring shortly after birth and it is, perhaps, not surprising that the efforts to reduce infant mortality should become increasingly successful as the infant advances in growth and development: the gains from a favourable environment are still limited by biological factors whether these are referable to a species or a particular post-natal age group.

An example of a biological determinant is the number of young born, and here the sow provides a good example. In this species, where the average number of piglets born per litter is 9 to 10, there is a premium on rapid development necessitating severe competition among the litter. This competition exerts its effect both before and after birth in that the litter is often uneven in size at term and, thereafter, the weak minority suffer. The plight of the weaker pigs is increased by the fact that the sow cannot attend to the needs of both weak and vigorous pigs simultaneously. The number of piglets born per litter also decrees that each new-born pig is small compared with the sow—a disadvantage in two respects for the sow must lie down to nurse while the baby pig must avoid being crushed by a mother weighing one to two hundred times its own weight. Yet, biologically, the sow might well afford these disadvantages, for even though she were to lose 80 per cent of her offspring she would still be replacing herself at a rate in excess of that for man and the other farm animals. There are other factors of a similar fundamental character that bear upon the question of comparative neonatal mortality but after assessing as far as possible

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their effects in the sow the mortality among new-born pigs seems so high that special conditions might be presumed to operate in this species.

There is no need to detail the many surveys that have shown and continue to show that pre-weaning mortality in the pig is commonly at a rate of 15 to 25 per cent (see Longwill, 1952). Such losses occur wherever pigs are reared, but little more can be learnt from these surveys (except that most of this mortality occurs within the first three days after birth) as the causes of death are commonly grouped under vague husbandry terms such as "bad doers", "chilling", "colds", "overlaying" or "scour". Hence, there is a need to study the question of piglet mortality as a whole (particularly as a problem in comparative medicine), to understand the interplay of physiological and pathological factors after birth and to attempt to clarify what is now a very confused syndrome—sickness in the new-born pig.

The work described below, undertaken as part of a wider study of the peculiar physiology and pathology of the new-born mammal, relates to one aspect of this problem only, but by integrating several previously unrelated observations it is hoped that it will lend more precision to future discussions on piglet mortality.

Materials and Methods

Sows and Litters.—In all, about 50 farrowings and, therefore, several hundred baby pigs, form the basis of these observations. Just over half of these were normal farrowings, being used to obtain physiological data, but within this group some adventitious pathological conditions were also observed. The remainder were farrowings in which pathological conditions were either reproduced experimentally or sought and studied in the field. In nearly every case parturition was attended by the writer, while the piglets were invariably studied in detail either continuously for 12 to 24 hours after birth or at frequent intervals for several days. The continuity of observation is stressed, for much is happening in the first 24 hours of a pig's life and the events of the first day or two largely determine the fortunes of a litter. When so much mortality is concentrated into 72 hours, a diagnosis based on only the final events presents special difficulties.

Blood Samples.—Baby pigs were bled by cutting off the end of the tail and mixed blood was allowed to drip freely over dried heparin. Moribund pigs were bled by cutting the throat vessels.

Blood Sugar Estimation.—Blood samples were deproteinised with sodium hydroxide and zinc sulphate or (later) with barium hydroxide and zinc sulphate. Reducing substances (taken as glucose in the absence of fructose) were estimated using the reagent of Somogyi (1945) but with a slightly modified procedure. Except for some of the early experiments, glucose standard solutions (checked by polarimetry) were included in each batch of estimations. The values for blood fructose concentration shown in Fig. 1 were obtained with the method outlined by the writer in 1952.

Liver Glycogen Estimation.—Pigs were killed for the estimation of liver glycogen. The whole liver was removed at once, cut into pieces and immersed

in hot 30 per cent potassium hydroxide. Glycogen content was estimated by the method of Seifter, Dayton, Novic and Muntwyler (1950).

The procedures for estimating glucose and glycogen have been discussed in detail elsewhere (Goodwin, 1954).

Experimental Studies

In 1941 Graham, Sampson and Hester reported that they had encountered among baby pigs in the field a condition of severe hypoglycæmia which they called "Baby Pig Disease". Later work from Illinois (Sampson, Hester and Graham, 1942) demonstrated that an indistinguishable syndrome could be produced by starving pigs shortly after birth in the laboratory. Thus it was apparent that the new-born pig is unable to refurnish glycogen and glucose from other available materials and while it remedies this acute deficiency in homeostasis by the second week of life (Hanwalt and Sampson, 1947), by then the critical neonatal period has been passed.

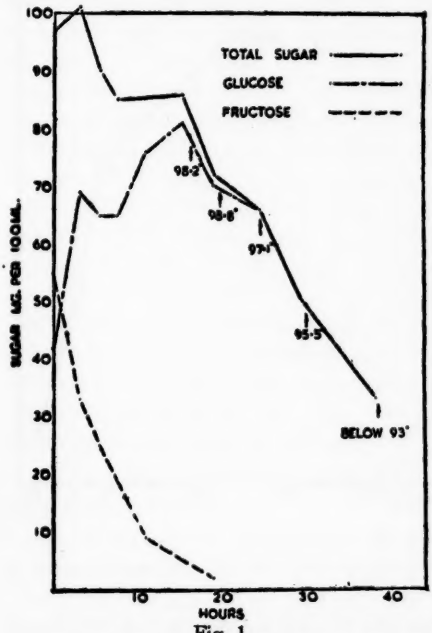

Fig. 1
Blood sugar concentration in a piglet starved from birth.

The above findings have two important applications: they bear directly on any study of carbohydrate metabolism in the new-born (Goodwin, 1954) and they are very pertinent to the broader problem of neonatal mortality. In the latter respect and as applied to the pig, they have been the source of much misinterpretation, particularly in Britain, where the terms "Baby Pig Disease" and "Hypoglycæmia" are used indiscriminately. This is unfortunate for the Illinois work, when placed in perspective, has made possible a greater understanding of the pattern of mortality in the baby pig.

That the blood glucose concentration declines on starvation in the new-born pig has been confirmed on many occasions in this laboratory and the figures below show the manner of this decline in two pigs. The first pig was denied milk from birth (Fig. 1) and although it demonstrated some initial regulation of the blood sugar, by about the 17th hour the decline of glucose concentration had started. The glucose concentration was allowed to decline until convulsions supervened at 39½ hours; then, within 15 minutes of sub-

cutaneous glucose therapy, the animal was on its feet and sucking milk normally. In pigs killed at delivery, the concentration of liver glycogen had been in the region of 10 to 14 per cent and this large store of glycogen is the probable foundation for the comparative stability of the blood sugar during the greater part of the first 24 hours.

The second pig (Fig. 2) remained with the sow until the 47th post-natal hour, when it was removed to the laboratory. The greatly increased resistance

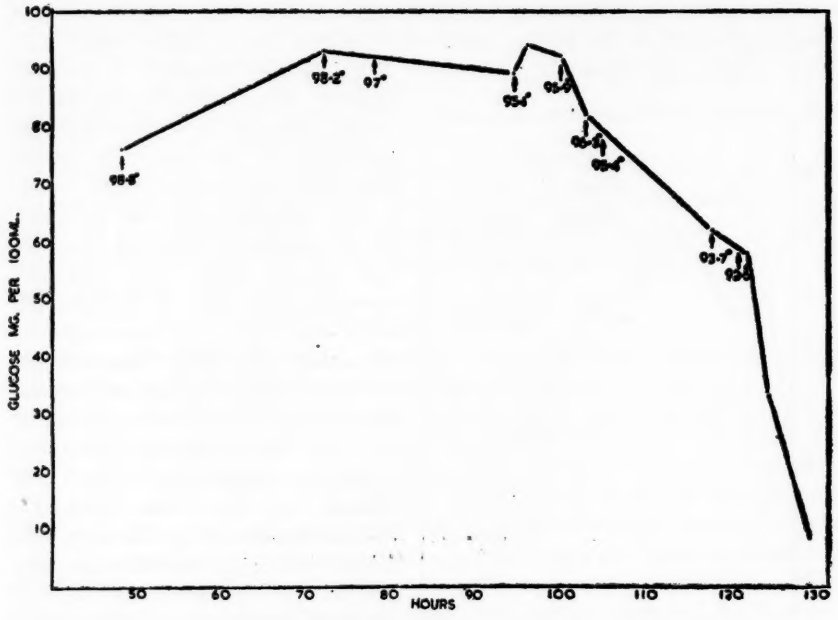

Fig. 2

Blood sugar concentration in a piglet starved from the 47th post-natal hour.

to starvation is very evident in this particular pig but the collapse of the blood glucose concentration eventually supervened as in the first pig. In both cases, the concurrent decline of rectal temperature is also shown. The time needed to develop a severe hypoglycæmia varies with other factors besides those of post-natal age (Hanawalt and Sampson, 1947) and environmental temperature (Morrill, 1952*b*) described hitherto.

Uncomplicated Hypoglycæmic Syndrome.

The syndrome of progressive hypoglycæmia, described here in general terms, is characteristic. The new-born pig (once recovered from the birth process and dry) is active, vigorous and strong on its feet, constantly grunting and in search

Note: As the time scales represent the duration of starvation, the latter is synonymous with age only in Fig. 1.

of food unless replete, and purposeful in behaviour. The skin is a reddish pink (when unpigmented) and very warm. Such a pig usually shows relatively little change in appearance until the blood glucose concentration has declined to about 50 mg. per 100 ml., but thereafter its activity and indeed its whole metabolism appears to run down in " clockwork " fashion and in step with the blood glucose. The progressive reduction in metabolism is evidenced most readily by this steady decrease in activity: at first the gait is uncertain and this deteriorates until the pig may maintain its balance only with the additional support provided by its nose on the ground or by resting on the carpi and straddling the hind legs. Later it may rest on its abdomen, but ultimately it falls on to its side and shows no further activity until the onset of convulsions. The latter are a common feature and although they may be incomplete (hesitant, frequently interrupted or reduced in extent) they are usually unmistakable. The pig exhibits strong and regular galloping movements of the forelegs with full extension during each cycle. Corresponding movements in the hind limbs are generally less extensive, the legs moving in a half-flexed position or being drawn up forward against the abdomen. During these movements, the head is drawn back slightly and the lower jaw champs rhythmically (typical air-hunger signs), developing a froth about the mouth. The period just preceding the first convulsion, together with the convulsion periods themselves, may be associated with a tortuous rigidity of the trunk and neck. Further signs of the declining metabolism are the fall in temperature (which can be appreciated by feeling the skin), increasing bradycardia (the heart rate can decrease from about 220 to 80 per minute or less between birth and the convulsion period) and the associated circulatory changes such as increasing skin pallor. Pigs in such straits, even when the blood glucose concentration registered as low as 7 mg.* per 100 ml., have been restored to normality following prompt glucose therapy.

From the experimental work at Illinois and in this laboratory two propositions were deduced:

I. Following the reduction of the high concentration of liver glycogen found at birth and before the establishment of satisfactory gluconeogenesis, a period of 3 to 4 days would occur in which the blood glucose concentration would be extremely unstable, fluctuating widely with the milk intake.

II. Under the conditions of I above, this peculiarity of carbohydrate metabolism could influence most of the morbidity of this period, directing a great variety of ætiological factors to be expressed in common symptoms and rendering fatal several conditions that could be surmounted in later life by the physiology of the adult.

Field Observations

The two propositions above were now tested clinically, but it is inevitable that observations of this type made in the field lack the precision associated with the controlled laboratory experiment. Furthermore, the collection of such data

* With existing methods and a single analysis, glucose concentrations as low as this can be only approximate.

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depends on both the availability of an ailing litter during the few critical hours before death and, at the same time, clear evidence of the primary pathological factor.

I. *General Instability of the Blood Glucose Concentration.*

The first proposition has been confirmed in a variety of ways. As the nutrition of the pig improves after birth, so does the blood glucose concentration steadily increase. Should the milk intake decline or suffer a temporary interruption, the concentration of blood glucose falls. Thus in a litter of pigs, a runt pig or a pig handicapped by an injury commonly has a blood glucose concentration well below that of the remainder of the litter and this concentration fluctuates about its low level according to the pig's success in obtaining milk. Similarly, during experimental work in which only some of a litter were removed frequently for examination, the pigs remaining on the sow grew more rapidly and also maintained their blood glucose at higher levels. The status of the blood glucose can thus be a sensitive indicator of the nutritional history which in turn summates the physiopathological experiences of the baby pig since birth.

An example of this relationship between the over-all milk intake and the blood glucose concentration has been well demonstrated in a litter of pigs suffering from hæmolytic disease. In this instance, some of the pigs were not permitted to suckle until up to 12 hours after birth. Those pigs left with the sow from birth experienced a rise in blood glucose concentration, but this was at the expense of a rapidly developing although non-fatal anæmia. The latter then interfered with normal nutrition, because although the affected pigs survived and gained well in weight, they were soon overtaken both in weight and blood glucose concentration by their litter-mates who had been withheld from suckling early on.

It was concluded, therefore, that although hypoglycæmia can result from complete starvation, it may more commonly result from a progressive reduction in milk intake. Under these conditions death may still be the result of hypoglycæmia, but it is postponed.

II. *Hypoglycæmia as a Secondary Clinical Symptom.*

The examples below have been chosen because, in each case, the primary ætiology was entirely or almost entirely clear. They serve only as illustrations and are not meant to be fully descriptive of any one condition, particularly as most of the primary ætiologies are well known. It is best to consider them under two main headings depending on whether the primary pathological process was in the sow or in the piglet.

Primary Pathological Process Affecting the Sow.

When so little is known of the causes and varying manifestations of agalactia in the sow, it is necessary to use descriptive terms that might apply to a number of different ætiologies or, indeed, have a common ætiology. The terms used here, " Total Agalactia " and " Post-parturient Agalactia ", are based on the main clinical difference between two conditions that are themselves responsible for two main forms of hypoglycæmia in the piglet.

1. *Total Agalactia.* In a pedigree herd of Large White pigs, whole litters

were dying on the second day of life. The time of death suggested complete starvation from birth and clinical examination of the litters confirmed the hypoglycæmic syndrome. Furthermore, the stomachs of the dead pigs were ballooned and, together with the lower bowel, completely devoid of milk. Bacterial cultures from the piglets were negative for pathogenic organisms. The sows exhibited few clinical signs (not even loss of appetite over farrowing) apart from complete agalactia.

Agalactia can sometimes be diagnosed falsely because the observer is unsuccessful in obtaining milk although the litter is fully nourished. In this herd, the diagnosis was confirmed in one sow at farrowing (a time when milk is usually abundant) and in another sow on the day after farrowing. In the second case, the litter was removed for about 2 hours, weighed and returned to the sow, where it was observed quietly from another box. The sow attempted to nurse her litter but the pigs were constantly trying different teats and did not gain in weight after remaining with the sow for about half an hour.

A variety of organisms were cultured from milk samples that were obtained after injections of posterior-pituitary extract and from vaginal secretions, but their significance was not clear. The condition did not respond to hormone or antibiotic therapy and the breeding stock had to be replaced. Under these conditions in the field, the hypoglycæmic syndrome is seen in its most acute form and without complications.

2. *Post-parturient Agalactia.* There are all degrees of post-parturient agalactia—a common one being that in which milk is abundant during farrowing but fades rapidly thereafter—but in this case the discussion will be limited to the type where lactation is initiated successfully and all appears to be well for a day or so *post-partum.* Hypoglycæmia in piglets has been seen following an acute mastitis in the sow in which the milk contained clots and yielded *Bacterium coli* on culture, but the case described in more detail below has features of special interest.

A litter of pigs was farrowed in which it was anticipated that hæmolytic disease of the new-born would develop. This disease was subsequently confirmed serologically, but it took a subclinical course. Regular estimations of hæmoglobin concentration in the piglets' blood showed that intravascular hæmolysis was slight and clinically the pigs were robust. It was surprising, therefore, when 5 out of the 7 pigs were found dead on the morning of the third day. Milk had been abundant at and after farrowing, and at *post-mortem* examination the stomachs contained milk curds. There was some evidence of overlaying and a laboratory diagnosis of crushing was made. However, a clinical examination of the two survivors showed that both were very weak, with hypothermia and bradycardia, and one showed air-hunger signs and commenced cyclic convulsions while being examined. The blood glucose concentration in this pig was estimated at 7 mg. per 100 ml., while in the other it was 30 mg. per 100 ml. and glucose injections restored both pigs to activity almost at once. They were subsequently reared artificially without further special therapy. This prompted an immediate visit to the sow, where complete agalactia was apparent both before and after

injections of posterior-pituitary extract. A few drops of milk obtained from one teat on the following day yielded hæmolytic streptococci.

This case demonstrates the difficulty of diagnosing the cause of death in piglets when presented only with the dead members of a litter. There were good reasons for two other diagnoses, for hæmolytic disease was undoubtedly present and almost certainly some pigs had been crushed—probably when they were weak from hypoglycæmia—but only a complete examination of the sow and surviving piglets allowed a correct assessment. It also demonstrates that hypoglycæmia as a cause of death may be accompanied by the presence of a curd in the stomach.

Primary Pathological Process Affecting the Piglet.

In the conditions described under this heading, hypoglycæmia results from pathological processes in the piglet that either completely prevent the piglet from sucking or create a degree of weakness such that the piglet cannot compete with its litter-mates or comply with the rigorous physical demands of the neonatal period.

1. *Bacterial Infections;* 2. *Myoclonia Congenita;* 3. *Hæmolytic Disease of the New-born.*

1. *Bacterial Infections.* Two conditions of septicæmia have been observed in the field in which the litters had to contend with the hypoglycæmic syndrome resulting from an interference with nutrition. In the first (a litter of 8), 2 pigs were dead by the 36th hour and 2 more were moribund. Here again the stomachs contained curds but, in this instance, milk was obtained readily from the sow. One of the moribund pigs was killed for bacteriological examination and yielded a pure culture of *Erysipelothrix rhusiopathiæ.* The following day the sow, together with a sow in an adjacent box, developed severe pyrexia and the characteristic skin lesions of erysipelas in the adult.

In the second condition, some pigs from a litter that was expected to suffer from hæmolytic disease were removed from the sow at birth and fed artificially for about 19 to 25 hours in an attempt to prevent their death from anæmia. They developed, however, a severe enteritis (probably from the unsatisfactory artificial diet, which was in any case over fed) and when returned to the sow, probably without the full protection of the colostral antibodies, they developed a rapidly fatal septicæmia. One pig had a blood glucose concentration of 29 mg. per 100 ml. (when it was too weak to stand) and was found dead 4½ hours later. A second pig also had a blood glucose concentration of 29 mg. per 100 ml., although it could just stand at this time: 5 hours later, when it was nearly dead, the concentration had fallen to 8 mg. per 100 ml. *Bacterium coli* was cultured from the viscera in 3 piglets examined and it was believed to be associated with the primary cause of the illness.

2. *Myoclonia Congenita.* This condition, so named by Kernkamp (1950), is well known. In many cases the violence of the muscular trembling is such that the young pigs are unable to suckle normally and develop a fatal hypoglycæmia. Other pigs in affected litters, while not so physically handicapped by

trembling, show no interest in feeding. As this condition is present at birth and it was the first condition investigated in this work, it seemed important to ensure that the trembling was not associated with some abnormality of carbohydrate metabolism, such as a deficiency of liver glycogen at birth. In a number of pigs slaughtered at birth, however, it was found that the liver glycogen and blood sugar concentrations were within the normal range.

3. *Hæmolytic Disease of the New-born.* Detailed results concerning this condition are being published separately (Goodwin, Hayward, Heard and Roberts) and it is sufficient to mention here that, whereas very severely affected pigs can die of anæmia a few hours before they would die of hypoglycæmia and many pigs are so slightly affected that their nutrition is uninterrupted, there is an intermediate degree of the disease in which either the interference with nutrition is undetectable clinically yet apparent in the blood glucose and weight gain figures, or in which the anæmia allows the development of a fatal hypoglycæmia. Often in the latter cases, the hypoglycæmia only advances the time of death for generally the pig would die of the anæmia itself in due course.

Theoretically, several other diseases of the new-born pig could result in death from hypoglycæmia but they have not yet been studied in this respect. Also, it is very probable that several diseases could kill the baby pig before the hypoglycæmia was severe enough of itself to result in death. Such conditions would need to be hyperacute such as the severest form of hæmolytic disease and possibly some septicæmias. However, certain conditions are seen in the first few hours of life that do not fit into any existing knowledge of the pathology of this period, and their development is too rapid for them to be related to the hypoglycæmic syndrome. In observing litters of pigs during the first twelve hours or so of life it is not uncommon to observe a pig to collapse before the time when the liver glycogen would be exhausted. Such pigs deteriorate very rapidly, often dying within an hour or so of first showing clinical symptoms. They quickly lose skin colour, become cold and limp while the blood becomes watery. There are no convulsions and the blood glucose concentration is often high. These cases have always been seen at a time when other observations were more urgent and as a result they have not been examined in detail. They may be related to the syndrome of post-natal anoxia in the human infant. A similar rapid collapse with hyperglycæmia has been seen in pigs delivered by Cæsarean section.

In concluding this section it must be mentioned that many deaths in baby pigs cannot be placed in any of the categories above. The most puzzling group now is that in which the piglets' stomachs contain curds and the sow supplies milk copiously on hand milking yet no bacterial or other primary pathological agent can be determined.

Discussion

The contention that hypoglycæmia would be a common and late symptom of many piglet diseases has been satisfied in all the distinct conditions so far examined. Some circumstances have been recorded, however, in which there

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was insufficient time for the development of hypoglycæmia. It is now necessary to examine the other diseases of the baby pig and determine the extent to which hypoglycæmia is a factor in their pathogenesis also. Morrill (1952*a*) suggested that in those conditions where hypoglycæmia exists and a curd is present in the stomach of the baby pig it would be useful to consider some inborn error of carbohydrate metabolism such as faulty absorption of sugar. It is the writer's view that such an approach is unnecessarily complex and several more conventional ætiological factors will undoubtedly be uncovered to explain the cases of this type where the ætiology is still unknown. The realisation of the widespread influence of hypoglycæmia in the new-born pig helps to reconcile the claims of those authors who explain the ætiology of hypoglycæmia or baby pig disease in different ways.

The common mould imposed on neonatal pathology by the physiology of the new-born has been illustrated with the symptom of hypoglycæmia and those consequences such as weakness, pallor, hypothermia, bradycardia and convulsions that are directly related. There are other symptoms, however, such as dehydration that will likewise be common to many conditions, and probably azotæmia will be found to be yet another symptom of this type. The extent to which these various symptoms are present will vary in different diseases, mainly according to the age of the piglet at death. Thus there is experimental evidence to support the idea that in diseases where death occurs early, hypoglycæmia would be especially important whereas when death is postponed, dehydration and azotæmia (Madsen, Earle, Heemstra and Miller, 1944; Morrill, 1952*b*) would be more obvious. There may well be other physiological factors in the new-born pig that have a similar general influence but there is one final cause of death and *post-mortem* finding—overlaying—that is so common that it will be mentioned in this context also.

The overlaying of many piglets under a variety of conditions has been observed during the course of this work. Although it is true that large, robust pigs are sometimes crushed purely by misadventure or a particularly clumsy sow, in general, crushing becomes more common as the strength of the piglet declines. There are two main causes of weakness in the new-born pig—small birth weight and disease. Those pigs that are small at birth are very liable to be crushed because their agility and muscular strength are poor, but pigs of all sizes at birth may be crushed eventually as sickness weakens them. The readiness with which hypoglycæmia develops in the new-born pig greatly facilitates these deaths by crushing, and the latter are so common that in experimental work it is frequently necessary to remove ailing pigs to the laboratory in order to observe the full course of the disease.

One of the main means of advance in medicine is to progress from the recording of symptoms to an understanding of ætiology and this progress is particularly desirable in the pathology of the new-born pig where the symptoms have been so confusing. It is suggested that a scheme such as is shown below could aid the understanding of this problem. The diagram is tentative and incomplete: it will require modification as new information becomes available

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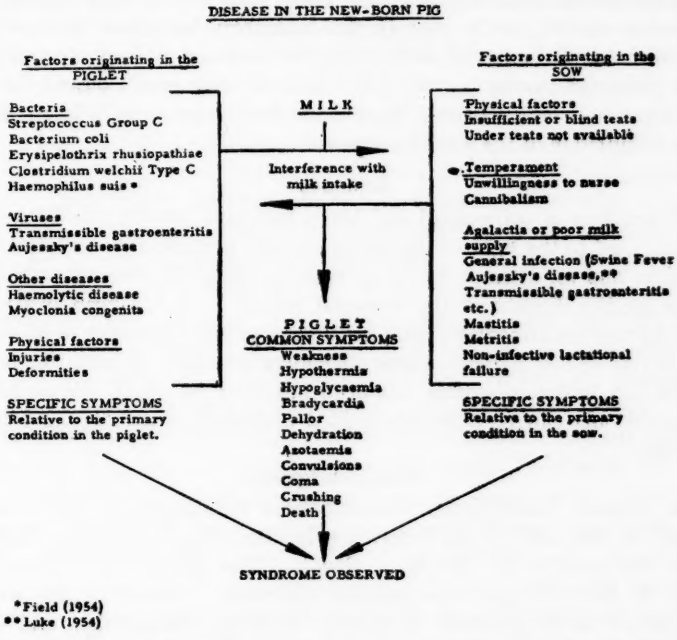
and the interrelations of other physiological factors are studied, but to think and work on these lines has particular advantages.

First, the multiple and complex pathology of this period becomes integrated into a reasonable pattern. Second, such an approach greatly assists clinical and laboratory diagnosis, for symptoms and laboratory findings can be apportioned to their respective ætiologies (specific conditions in the sow and piglet, or secondary physiological factors) and the critical points can then be isolated



*Field (1954)
**Luke (1954)

upon which a specific diagnosis depends. Third, a rational basis is provided for therapy and preventive husbandry. As a therapeutic example, it is equally undesirable to treat agalactia in the sow without fortifying the litter with glucose as it is to treat hypoglycæmia with glucose without recognising and attending to any primary condition in the piglet. Therapy, however, is particularly difficult in the baby pig because the diseases develop so rapidly and diagnosis is difficult in the field; hence preventive husbandry is more important. The observations of Morrill (1952*b*), where the survival time of pigs starved from birth was increased from 28 to 84 hours merely by increasing the ambient temperature from 59° to 88° F., provide a good example for preventive husbandry and indicate one reason for the use of infra-red lamps. A low environmental temperature, therefore, can greatly aggravate the difficulties of the ailing piglet but it would be preferable if the term " chilling " were not used to describe these circumstances. True chilling has been reported by Taylor, Orem, Doyle and Vestal (1952) and is not likely to be widespread in this country.

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It is apparent that the new-born pig must contend with disease in the shackles of its homeostatic deficiencies and it is important to know whether similar handicaps are shared by the new-born of other species. It is possible that the inability to withstand a short period of starvation may be peculiar to the baby pig; on the other hand, the new-born of other mammals might experience difficulties in other forms. A start has been made already in the examination of other species for their ability to resist starvation. Sampson, Taylor and Smith (1955) have shown that the new-born lamb does not develop hypoglycæmia rapidly when starved, while work in this laboratory has shown that calves can be kept without all food and water from the moment of birth (provided that they are protected by the injection of a little of their dam's serum) for over a week and yet show little concern. Such calves have been reared without difficulty from the completion of the starvation period.

Summary

The problem of widespread and heavy mortality among new-born pigs has been considered in the light of the biological and physiological factors operating at this time and an attempt has been made to integrate these general findings with the specific diseases of the neonatal period. It has been demonstrated that the former fundamental principles direct separate ætiologies to be expressed largely as one disease pattern, which engenders confusion in both diagnosis and treatment. A tentative scheme has been proposed by which the pathology of the new-born pig can be both sub-divided according to ætiology and still seen as a whole.

Acknowledgments

The routine bacteriological examinations mentioned in this work were performed by the staff of the Veterinary Investigation Laboratory, Cambridge, through the courtesy of Mr H. I. Field. The author is indebted to Mr P. J. D. V. Brett for part-time general assistance. Much of this work was carried out in the Department of Animal Pathology, Cambridge, and the later investigations have been aided by a financial grant from the Agricultural Research Council.

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FELINE ENTERITIS IN THE LYNX, THE CHEETAH AND OTHER WILD FELIDÆ

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Records of the incidence of feline enteritis among the wild felidæ are both uncommon and unsatisfactory. It is significant that all such reports concern wild members of the cat family living in captivity; and, in spite of inquiries to game wardens and others, reliable observations of the disease are unknown among animals at liberty in their natural habitat. It is probable that the disease is normally confined to the domestic cat, and only occurs among wild animals under certain abnormal conditions. Although the wild felidæ may, in general, be highly susceptible to the disease, exposure to infection is extremely rare due to the fact that they do not live the gregarious existence of their domesticated relatives. Thus, feline enteritis manifests itself among the wild "cats" only when they are brought into close contact with one another and with the domestic cat and his infected excretions and discharges. The city zoological gardens present an unequalled environment for the spread of the disease.

Some confusion, if only of popular terminology, exists in the older literature between "feline influenza" on the one hand and on the other the "feline enteritis," "infectious feline agranulocytosis" and "malignant panleucopænia of cats" which were regarded by Jennings (1949) as probably identical. In some instances both groups are recorded, regardless of symptoms, as "cat distemper."

An early reference to infection in the wild felidæ, by Hindle and Findlay (1932), recorded that "feline distemper" has been seen in the American and African tiger-cats (*Felis tigrina* and *F. aurata*), the caracal, the ocelot and the leopard. At that time, there were no records of infection in the larger members of the family—lions, tigers, pumas—though young leopards were known to be susceptible. The subsequent description of the symptoms and the pathological changes suggests that at least some of the cases may, however, have been the condition now recognised as "feline influenza."

For the same reason the earlier records of feline enteritis at the Gardens of the Zoological Society of London, quoted by Cockburn (1947) may be suspect. Here tigers, leopards, wild cats, lynxes, servals, leopard cats, tiger cats, ocelots, eyra cats and cheetahs are all recorded as casualties, though lions appeared to be immune. Only one doubtful case occurred among the civets and genets. Nevertheless, Cockburn actually observed the condition of "cat distemper" (infectious enteritis) in leopards, wild cats, cheetahs and an ocelot—and attributed sudden overnight death in a lynx and a further leopard to this disease. The white-cell counts of two of the cheetahs were given as ten thousand per c.mm., but it should be remembered, however, that whilst the "normal" count for the cheetah is unknown, this figure, according to Jennings (1947), would not be grossly abnormal for the cat.

Goss (1948), in discussing the evolutionary standpoint, traced a paleo-

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genetic susceptibility in the felidæ and recorded that the disease has been seen in "practically all the cats in the New York Zoological Park." Unfortunately, the particular species infected are not specified. Cases in the leopard, serval, lynx and lion alleged to have occurred in the Sudan were cited in an addendum. Goss also reported the disease in a raccoon which, oddly enough, belongs to the family Procyonidæ. Grosso and Rodriguez (1948) diagnosed the disease in the jaguar, puma, ocelot, mountain cat, eyra cat, lynx and lion; and have successfully transmitted the disease from the jaguar to the domestic cat. McGaughey (1950) observed feline enteritis among young leopards at the Zoological Gardens, Dehiwala, Colombo, and transmitted it to cats by intranasal instillation and subcutaneous inoculation. Contact was shown to occur between these leopards and mongrel cats from a district in which the disease is enzootic.

In the past, diagnosis of the disease in the wild felidæ appears to have been largely based on clinical symptoms and on occasional post-mortem examination. In only one case (McGaughey, 1950) has the identity of the disease in both species been supported by known contact infection between the domestic and wild "cat," and then confirmed by passage back to the first species. Grosso and Rodriguez did fulfil the second requirement by transmitting the virus to the domestic cat from the jaguar.

The following account relates the circumstances of transmission of fatal infection from the domestic cat to the lynx, thence to the cheetah and, finally, again to the domestic cat.

The Lynx and the Cheetah—Case Reports

The first case to be seen in this series was a young lynx of about two months old, the property of a "white hunter" and animal dealer. The animal had been vomiting at short intervals for about twenty-four hours and was completely prostrated. The visible mucous membranes were pallid, respirations rapid, and the fur was matted with yellowish vomitus. The temperature was 98.3° F. Prior treatment had consisted of penicillin injections and glucose saline given orally. Parenteral administration of the glucose saline was advised and chloramphenicol prescribed, but the lynx died within a few hours.

Two litter mates of this animal were apparently healthy. These were removed to other quarters several miles away and there isolated. Treatment with chloramphenicol was recommended but was not carried out. Four days later both animals developed clinical signs of the disease and died on the sixth and eighth days respectively.

The signs usually associated with feline enteritis were present on post-mortem examination of the first lynx. The stomach and intestines were empty except for a yellowish catarrhal exudate. The terminal part of the duodenum showed marked congestion of both the mesenteric vessels and the mucosal walls. The lymph nodes of the abdominal cavity were swollen and œdematous. Slight congestion was present in the kidney tissue and in the meningeal vessels. A few small patches of early pneumonia occurred in both lungs. The liver appeared

fatty. Blood smears, although made very soon after death, showed an almost complete absence of granulocytes.

Ten days later an eight-month-old cheetah, kept on the original premises, refused all food and vomited a yellowish frothy material at frequent intervals. The same treatment was prescribed but, due to the ferocity of the animal in the early stages at least, this was difficult to carry out. After five days the cheetah died, having consistently refused food and water and manifesting signs of intense abdominal pain since the onset of symptoms. The post-mortem findings were identical with those in the case of the lynx, except that the lungs and the meninges were macroscopically normal and the spleen somewhat swollen.

Spleen tissue, taken from the cheetah under reasonably aseptic precautions, was ground up and emulsified in normal saline. Part was freeze-dried and sealed off *in vacuo*, the remainder being stored in a sterile tube at 4° C.

Two healthy semi-wild six-week-old mongrel kittens of the same litter were obtained from a locality free from the disease, and inoculated subcutaneously with 1.0 ml. of reconstituted dried spleen (cat 1) and with 1.0 ml. of "wet" material of five days' storage (cat 2). Cultures from both these inocula remained bacteriologically sterile. Differential white-cell counts were made at intervals on the blood of these cats, which were kept separately in isolation.

On the second day after inoculation both cats showed raised temperatures and were reluctant to eat; whilst on the following day the temperatures of both were subnormal, vomiting occurred frequently and anorexia was complete. Cat 1 developed a slight neutrophilia, then a progressive neutropænia with a relative lymphocytosis, and died on the fourth day after inoculation—apparently without having eaten since the second day. Cat 2 showed a transient neutrophilia, with a marked "shift to the left" in the neutrophil count, followed by a neutropænia less severe than that of cat 1. At about the sixth day the temperature became approximately normal. At this period the animal adopted a peculiar crouching attitude and a coarse tremor of the head and neck was apparent. Diarrhœa developed. Later the temperature fell below normal, and the animal died during the eighth night after inoculation.

At autopsy the classical changes of feline enteritis were present in both cats —empty stomach and intestines, slight congestion of the mucosa of the small bowel which contained a yellow-stained catarrhal exudate, enlarged œdematous abdominal lymph nodes, slight enlargement of the spleen and fatty change in the liver. The red marrow of the femur was abnormally soft, and a stained smear of this showed an almost complete absence of the later myelocytes. Apart from the congestion of the alimentary mucosa already mentioned, histological sections failed to reveal anything significant. Intranuclear inclusion bodies were not definitely identified. Only a few colonies of coliform organisms grew in cultures of the spleen and femur of cats 1 and 2.

The uninoculated litter-mates of cats 1 and 2 were left at liberty and

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remained healthy during the period of observation. Rabbits, guinea-pigs and mice proved insusceptible to infection.

Animal	Criteria of Infection	REACTIONS Days Post Inoculation 1	2	3	4	5	6	7	8	9	Normal Jennings 1947
	White blood cells	%	%	%	%	%	%	%	%	%	
Cat 1	Neutrophils Non-segmentors	10.0	-	4.0	6.0						N.A.
	Multi-lobular	60.0	-	20.0	14.0						N.A.
	Total	70.0	-	24.0	20.0						56.7 ± 6.7
	Eosinophils	4.0	-	2.0	7.0						5.2 ± 2.1
	Lymphocytes	23.5	-	63.0	51.0						32.9 ± 7.3
	Monocytes	2.5	-	11.0	22.0						4.5 ± 1.7
Cat 2	Neutrophils Non-segmentors	12.0	-	42.0	32.0	-	16.5	-	14.0	-	N.A.
	Multi-lobular	42.0	-	38.0	28.0	-	28.5	-	45.0		N.A.
	Total	54.0	-	80.0	60.0	-	65.0	-	59.0		56.7 ± 6.7
	Eosinophils	4.0	-	3.0	3.0	-	3.0	-	2.0		5.7 ± 2.1
	Lymphocytes	34.0	-	15.0	31.0	-	47.0	-	36.0		32.9 ± 7.3
	Monocytes	8.0	-	2.0	6.0	-	5.0	-	3.0		4.5 ± 1.7
	Temperature	°F	°F	°F	°F	°F	°F	°F	°F	°F	
Cat 1		101.0	104.1	99.5	99.0	Dead					
Cat 2		100.6	105.5	98.1	97.3	100.2	100.0	98.3	97.8	Dead	

%-figures are mean of 600 cells, corrected to nearest 0.5%.

Discussion

Very few structures resembling eosinophilic intranuclear inclusion bodies were observed in sections of the small intestine stained, as recommended by Jennings (1949), by Goodpasture's method. This absence was to be anticipated, perhaps, since both cats inconsiderately died during the night and so were not autopsied until morning.

The naturally infected lynx and cheetah developed pathological changes which were remarkably consistent with one another and with those found in the artificially infected cats.

The difference of survival time between the two cats is inexplicable. Possibly it may be due to a higher live virus titre in the freeze-dried material than in that stored at c.4° C. for five days, but it is probable that the difference can be accounted for by variation in individual susceptibility—even between two animals of the same litter.

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Further inquiries revealed that the disease had first occurred on the infected premises some five months previously. At this time a young adult pet tomcat had exhibited clinical signs of the disease after a series of nocturnal excursions, but had subsequently recovered. About twenty-one days later a young lynx (lynx 1) of about six months old, had become infected and died after a somewhat protracted illness. Wild felidæ had not been kept on the premises for some three months before the first lynx seen by the writer (lynx 2). This became infected soon after its arrival. Both these animals, being very young, were allowed freely into the house. Spread of the infection was thus by direct contact from the tomcat to lynx 1, via fomites from this to lynx 2, by direct contact to its litter-mates (lynxes 3 and 4), from these via fomites to the cheetah and thence by parenteral injection to cats 1 and 2 in the laboratory.

Summary

1. Reports of feline enteritis in wild felidæ are reviewed, the following "cats" being recorded as susceptible although some confusion with feline influenza may have occurred in the earlier reports: Caracal, cheetah, eyra cat, jaguar, leopard, leopard cat, lion, lynx, mountain cat, ocelot, puma, serval, tiger, tiger cats (American and African) and wild cats.

The disease is recorded in the raccoon.

2. An account is given of the transmission of feline enteritis from a domestic cat to young lynxes, thence to a cheetah and, finally, experimentally to two cats whose reactions to the disease are noted. The epizootiology of the outbreak is discussed briefly.

3. Constant symptoms in the cat, cheetah and lynx were anorexia, yellowish vomitus, abdominal pain, depression, collapse and death. These were associated with a marked drop in numbers of white blood cells of the granular series.

Acknowledgments

My thanks are due to Mr. M. L. Burdin, B.Sc., M.R.C.V.S., for the preparation of histopathological sections; and to the Director of Veterinary Services, Kenya, for permission to publish the foregoing.

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AN IMPROVED METHOD FOR THE PREPARATION OF AN ANTIGEN FOR THE RAPID SLIDE TEST FOR CONTAGIOUS BOVINE PLEURO-PNEUMONIA*

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Introduction

SINCE the first description of a whole-blood rapid slide test for Bovine Contagious Pleuro-pneumonia (Newing and Field, 1953) several improvements have been made in the method of preparation of the antigen. These are:—

(a) Methods giving improved culture yields. Ordinary liquid media cultures of the Pleuro-pneumonia organism contain a very small amount of bacterial bodies, even after prolonged incubation. Culture methods giving higher yields than those previously used were required if the rapid slide test was to become economically feasible.

(b) Improvements to the stability of the antigen. Some samples of antigen tended to auto-agglutinate with normal sera, also the antigen particles tended to conglomerate on keeping. Some variation from batch to batch of the agglutinability of the antigen was also experienced.

Improvement of Culture Yield

Improvement in the growth of the pleuro-pneumonia organism in shallow liquid cultures, compared to that in deeper layers, was noted by Campbell and Turner (1953). Later, Rodwell and Rodwell (1953) observed that growth was stimulated markedly by aeration. The increase of growth rate and yield of some bacteria by aeration has been recognised for some time, and was reported by Roux and Yersin in 1890 for the diphtheria bacillus. Methods for aerating cultures of many organisms have been developed. Usually two methods have been employed: (i) Shaking thin layers of culture in Erlenmeyer flasks and (ii) Bubbling air through the medium (Sparger aeration).

The medium is a modification of that used by Edward (1947) for the cultivation of pleuro-pneumonia like organisms. A beef infusion broth is supplemented by the addition of 2 per cent peptone, 10 per cent horse or ox serum, 10 per cent yeast extract, and 0.1 per cent glucose. The *pH* is adjusted to 7.4 and the medium sterilised by filtration directly into the culture flasks, each flask containing 1.5 litres. To reduce the possibility of contamination, one part in 6,000 w/v of thallium acetate is incorporated in the medium together with 200 units per ml. of sodium penicillin. The penicillin is added in two portions, one during filtration into the culture flasks, and the other after incubation but before inoculation.

Three-litre boiling flasks were used as culture vessels. Aeration was provided by a glass tube, with the end drawn down to a capillary point and projecting into the medium (see diagram I). A cotton wool filter was attached directly to the top of the "sparger" tube. The filter consisted of a 10 cm. length of 3 cm.

* Received for publication, October, 1954.

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bore " Pyrex " glass tubing, closed by a single hole rubber bung at each end, and packed tightly with absorbent cotton wool. The cotton wool packing had been treated previously by soaking in 1 : 1,000 alcoholic acriflavine solution, drying, soaking in aqueous saturated mercuric chloride solution and finally drying in a hot air oven. These bactericidal and fungicidal agents prevented contaminants growing through the filter, and being non volatile were not carried through into the medium.

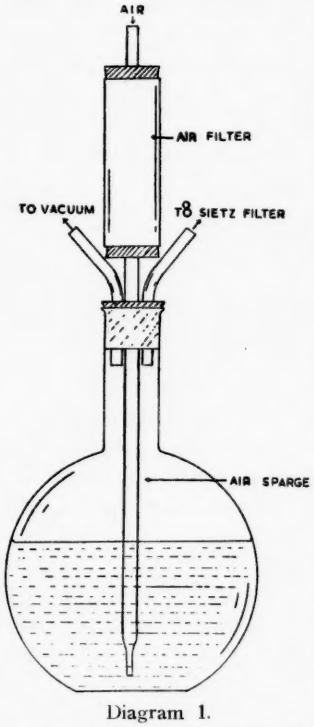

Diagram 1.
Apparatus for aerated culture of the pleuro-pneumonia organism.

Air was supplied to the top connection of the filter by means of a rubber connection from a manifold maintained at a constant air pressure of 5 lb./sq. in. by means of a reducing valve from a supply at 50 lb./sq. in.

In these experiments no diffusers were used on the sparger tubes, adequate aeration being obtained without their use. Two short glass tubes were fitted to the top of the culture flasks to provide connections for the Seitz filter used in filling the vessel, and for vacuum connection. During the culture period these tubes provided an exit for the air, and were covered by short glass tubes containing cotton plugs. The complete flasks are sterilised by autoclaving at 15 lb./sq. in. for 30 minutes.

After a 48-hour check incubation, the flasks were inoculated with 10 ml. of a 7-day Martin's broth culture of the KH3/J strain of pleuro-pneumonia organisms; a 10 ml. hypodermic syringe with a 4 in. needle being inserted down one of the short tubes.

During the early stages of this work a half-inch layer of sterile liquid paraffin was delivered on top of the medium to control frothing. This was not effective, and led to difficulties during harvesting; it was abandoned. Later batches of antigen were produced without the aid of a froth-control agent, the rate of aeration being adjusted to give the maximum tolerable degree of frothing. The low rate of aeration so obtained gave adequate growth, but small fluctuations in the air pressure were liable to cause excess frothing, with an overflow from the flask and resultant contamination. In order to eliminate excess foaming, a sample of " Avlinox "* I.C.I. (Polyoxy-ethylene derivative of ricinoleic acid) was obtained for trial as an antifoaming agent. The effect on growth was ascertained by tube cultures. After six days' incubation the opacities of the tubes were compared with uninoculated controls by means of a Hilger " Spekker " photo-electric absorptiometer, and viable counts obtained by triplicate serial

*Obtained by courtesy of Messrs. Imperial Chemical (Pharmaceuticals), Ltd., Manchester.

dilutions. Concentrations of up to 1 : 50,000 v/v of "Avlinox" did not depress growth to any detectable extent and if the concentration was raised to 1 : 5,000 the viable count was merely reduced from 10^9 per ml. to 10^7 per ml. The results are shown in Table 1.

TABLE 1

Effect of "Avlinox" on growth of pleuro-pneumonia organism

"Avlinox" concentration	Average absorptiometer reading.	Approximate viable count.
Nil	0.043	1 in 10^{-9} ml.
1:100,000	0.045	1 in 10^{-9} ml.
1: 50,000	0.043	1 in 10^{-9} ml.
1 :10,000	0.040	1 in 10^{-7} ml.
1 : 5,000	0.037	1 in 10^{-7} ml.

For a preliminary trial "Avlinox" was incorporated in the medium by adding a suitable quantity of 1 per cent emulsion to the empty flasks before sterilisation. Two batches of three flasks each were prepared, using 1 : 50,000 "Avlinox," and antigens prepared from each; both antigens were agglutinated readily with positive and negative sera to a titre of 1 : 64; further tests were made against dilutions of horse serum and they were agglutinated readily.

From these results it appears that "Avlinox" alters the surface charge of the antigen particles to such an extent that they are non-specifically agglutinated by normal serum. All subsequent batches of antigen were produced without the aid of a froth depressant.

Culture Yield

Owing to the considerable demand for antigen, and the limited culture facilities available, no growth curves under aerated conditions were compiled. An arbitrary culture period of six days' incubation was utilised as this gave a reasonable yield and permitted a weekly cycle of operations. The cultures were harvested as detailed elsewhere in this paper, the nitrogen content of the separated organisms was estimated by a micro-Kjeldahl technique, and dry weights were estimated after desiccation to constant weight over phosphorus pentoxide under vacuum. The original volume of culture medium of each batch was measured at the time of harvesting, and from these figures the yield of bacterial nitrogen and bacterial dry weight per ml. culture were calculated. The results obtained from six batches of culture are given in Table II. Control batches of non-aerated culture were grown in similar flasks, the stopper and

aeration tube being replaced by a cotton-wool plug. These results are also shown in Table II.

There is a considerable increase in yield from aerated cultures when compared with that from non-aerated ones. The average ratio of nitrogen yields of aerated compared to non-aerated culture is 3.02, with a maximum of 3.4. The percentages of bacterial nitrogen or dry weight in both the aerated and non-aerated cultures were similar, i.e., 6.62 per cent and 6.13 per cent respectively. On the basis of nitrogen yields, the amount of antigen produced, standardised to contain 1.5 mgm.N/ml., would be 14.5 ml./litre from the aerated culture and 4.8 ml./litre from the non-aerated culture.

TABLE II.

Comparative yields of aerated and non-aerated cultures

	AERATED			NON-AERATED		
Batch No.	mgm.N/ml culture	mgm.dry wt./ml. culture	%N of D.W.	mgm.N/ml culture	mgm.dry wt./ml. culture	%N of D.W.
I	0.0184 mgm	0.318 mgm	5.8%	0.00785	0.1230	6.4%
II	0.0216	0.250	8.6%	0.00642	0.1695	5.9%
III	0.0187	0.346	5.4%	0.00821	0.1765	6.9%
IV	0.0196	0.330	5.95%	0.00674	0.1160	5.8%
V	0.0245	0.356	6.9%	0.00749	0.1375	5.45%
VI	0.0237	0.334	7.1%	0.00692	0.1100	6.3%
Average	0.0218	0.323	6.62%	0.00724	0.1387	6.13%

D.W. = dry weight

Preparation of the Antigen

The method of preparation of the antigen differs in several respects from that already described (Newing and Field, 1953). For the sake of clarity the process is described again. After 6 days' incubation at 37° C. aerated culture flasks are checked for contamination by dark ground microscopical examination of a loopful of material.

The contents of uncontaminated flasks are passed through a high-speed continuous flow centrifuge at a rate of approximately 25 to 30 ml./min. A modified Lister milk separator with a 4 in. diameter bowl rotating at 14,000 r.p.m. is used as the centrifugal separator, and at this speed removes all the organisms from the culture. After all the culture has passed through the separator, the medium remaining in the bowl is displaced by 1 litre of normal saline which is passed through at the same rate. The organisms are then harvested as a sludge from the inside of the separator bowl and re-suspended in the liquid left in the bowl. This suspension is heated for 30 minutes in

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a water bath at 100° C. This substrata is satisfactory whereas distilled water is not. Distilled water suspensions behave irregularly, some samples being completely non-agglutinable by specific anti-serum. Priestley (1952) refers to difficulties experienced when attempting to use the complement fixation antigen of Campbell and Turner (1953) for a slide agglutination test, this having been prepared by heating a distilled water suspension of organisms. In order to overcome irregular agglutinability Priestley treated the antigen, before use, with a dilute solution of sodium hydroxide. In my experience alkali treatment of the antigen renders it agglutinable, but often destroys its specificity. Sodium hydroxide concentrations in the order of 1 : 4,000 w/v slowly dissolve the antigen particles. On neutralising with dilute hydrochloric acid a precipitate is formed, which is specifically agglutinated by pleuro-pneumonia anti-serum. The density of the re-precipitated antigen is considerably less than that of the original. Alkali treatment of the antigen has been found unnecessary when the organisms are heated as normal saline suspension, all batches produced in this way being specifically agglutinable.

On heating, the suspension of organisms undergoes some degree of coagulation. After cooling, the coagulum is separated by centrifugation at 4,000 r.p.m. for 30 minutes, and the supernate discarded. The coagulum is re-suspended in normal saline to a creamy consistency. Duplicate samples of 0.25 ml. of the suspension are taken and the nitrogen content estimated by a micro-Kjeldahl determination. The concentrated suspension is diluted to contain 1.5 mgm. Nitrogen per ml. This concentration has been arbitrarily fixed, as it gives a sufficient density for the test to be read easily in the presence of whole blood. The suspension is homogenised by passing it three times through a " Premeier " colloid mill, with the cones set to a gap of 0.003 in. To aid in maintaining a homogeneous suspension, " Shell " Teepol is added to give a concentration of 1 : 500 w/v. The suspension is stained to the requisite colour by the addition of an aqueous solution of Methyl violet. Sodium citrate 0.3 per cent w/v is added to act as an anti-coagulant. The antigen is preserved by the addition of 1 :5,000 w/v Merthiolate. Earlier samples were preserved by the addition of 0.5 per cent phenol, but it was found that this substance was responsible for the aggregation of the antigen particles.

In order to determine the stability of the antigen when preserved with Merthiolate, two samples of one batch were stored at 4° C. and 37° C. respectively. At weekly intervals serial dilutions were prepared from dried positive and negative sera. One drop of antigen was then added to one drop of each serum dilution on a white tile. both reagents being measured with Dreyer's dropping pipettes. End points were read as the last dilution showing any detectable agglutination one minute after mixing. The pools of antigen serum mixture were agitated by rocking the tile. The end points were well defined, and no agglutination to higher titres occurred after standing for periods longer than one minute.

Both samples agglutinated readily to a dilution of 1 : 64 with the positive serum, and not at all with the negative serum. At the time of writing, after

a period of 12 weeks, no decrease in titre has been observed in either antigen, and neither shows any tendency to agglutinate with the negative serum.

Discussion

The use of aeration techniques for the cultivation of the pleuro-pneumonia organism provides a simple means of obtaining high yields of that organism, which normally gives very poor yields when grown in broth. No attempt has been made to explain the mechanism of the increased growth, whether it is due to an increase in oxygen tension, removal of carbon dioxide or the effects of mechanical agitation. The earlier observation by Turner (1935), that the organism behaved as a micro-aerophil, would not seem to be borne out by these experiments, nor by the later observations of Campbell and Turner (1953).

The low rates of aeration obtainable without the use of anti-foaming agents did not appear to be disadvantageous, as the increased growth obtained was of a similar order to that obtained by other workers in this field. In the apparatus described, no means of diffusing the air bubbles throughout the medium was provided as this tended to increase the amount of froth produced, although Gee and Gerharde (1946) reported that the use of a sintered carbon diffuser reduced considerably the amount of air required to produce a given yield of Brucella.

The effect of the anti-foaming agent "Avlinox" on the specificity of the antigen is unfortunate as this substance provides a most effective method of foam control. The use of inert foam depressants, such as lard, on the surface of the medium was rejected owing to the increased difficulty of harvesting and of cleaning glassware. The culture medium used in this work is only slightly modified from that described by Edward (1947). It was found that this medium, as originally prepared, tended to turn alkaline during the culture period, thus prematurely arresting growth. The inclusion of a small amount of fermentable carbohydrate, in the form of glucose, prevented this trouble.

The method of preparation of the antigen varies in several respects from that first described owing to the occurrence of auto-agglutination and the failure of some batches to be agglutinated satisfactorily. Unheated saline suspensions of the organism can be agglutinated readily by the specific anti-serum, but this property is soon lost on standing, presumably due to autolytic changes. Heating of the organisms immediately after harvesting fixes the agglutinating properties of the organism, and causes larger flocculation. If the suspension of organisms is allowed to stand until the power of specific agglutination is lost, it cannot be restored by heating. Other fixative agents, such as formalin, have been tried but none act as satisfactorily as heat. The period of heating in the water bath has been increased from 10 minutes, as first described, to 30 minutes, as this reduces the tendency to auto-agglutination. Since the method of heating the organisms as a saline suspension has been adopted, no instances of non-agglutinability of the antigen have occurred. Some of the samples of antigen which were unsatisfactory for the agglutination test behaved perfectly satisfactorily as complement fixing antigens. Conversely, heated saline suspensions

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failed to fix complement in the presence of the specific anti-serum. This suggests that two antigen/antibody systems may be concerned.

The previously described methods of standardising the antigen were unsatisfactory owing to variation in the size of the antigen particles. Standardisation by nitrogen content has been adopted as the procedure is fairly simple, and using a micro-Kjeldahl method only a very small sample of antigen is required. Campbell and Turner suggested that this method might be more suitable for standardising their complement fixing antigen than the opacity method previously used.

The homogenisation of the antigen by means of a colloid mill produces a far more stable antigen suspension than the earlier method of grinding in a ball mill, and the suspensions so produced are free from any large particles. The addition of "Teepol" aids in the maintenance of an even suspension and does not appear to interfere with the flocculation reaction. It is intended to investigate the effect of other suspending agents, as these become available.

The stability of the antigen in its present form, despite prolonged exposure to temperatures as high as 37° C., is likely to be of great value in the field, particularly in the more inaccessible parts of the country.

Summary

A method of obtaining increased culture yields of the causal organism of contagious bovine pleuro-pneumonia by means of aerated cultures is described. The yields obtained from aerated cultures are in the order of three times those obtained from non-aerated cultures, using the same medium and measured on the basis of bacterial nitrogen yield.

The use of a surface active agent to control frothing in the culture flasks was investigated, and although this agent enabled greater rates of aeration to be used, it interfered with the specificity of the resulting antigen. The details of the method of preparation of a rapid slide test antigen for use in diagnosing contagious bovine pleuro-pneumonia are given. The method varies in several respects from that described previously.

Acknowledgments

I wish to thank Mr. J. R. Hudson for drawing my attention to the use of aeration culture techniques and Mr. R. M. Gambles, Senior Veterinary Research Officer, for comments on the use of the antigen for the complement fixation test.

I am grateful to the Inspector General of Animal Health Services, Nigeria, for permission to publish this paper.

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TOXICITY STUDIES ON SODIUM COPPER CHLOROPHYLLIN

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THE complete absence of any harmful effects from the administration by various routes of water-soluble chlorophyll derivatives to different mammals has been reported by American workers (Chamelin, 1942; Hyams and Helfert, 1942; Smith, 1944; Rakieton and Newman, 1949). Their findings have been confirmed and extended in the present study, which has been conducted with solutions of a derivative that is now available in Great Britain for therapeutic purposes in man, and which has recently been shown to be capable of producing "green yolks" in laying birds and potentiation of growth in cockerels up to 12 weeks of age (Worden, Bunyan and Davies, 1955*a*, *b*).

Experimental

The preparation employed was sodium copper chlorophyllin 100 per cent (Council, 1951), which for oral administration was packed in gelatin capsules of suitable size and for parenteral administration was injected as a sterile 2 per cent w/v solution in distilled water adjusted to *pH* 7.4. Since the results were consistently negative, they may conveniently be expressed in tabular form (see Tables I and II). The results in Table I include some that were obtained during the course of the studies on the experimental production of "green yolks" (Worden *et al.*, 1955*a*, *b*). The passage of small quantities of sodium copper chlorophyllin into the yolk of eggs produced by laying birds, and intense coloration of the fæces were in fact the only detectable effects in intact animals. The distribution of sodium copper chlorophyllin in the tissues of animals sacrificed following the administration of this substance is being further reinvestigated, but in no instance has any pathological change been detected, despite extensive histological examination.

The only untoward effect noted throughout these investigations was temporary malaise in one pig subjected to the intraperitoneal injection of 100 ml. of the solution. In this instance considerable struggling occurred when the animal temporarily escaped restraint during the injection and the peritoneum probably suffered torsion or mild bruising. The animal had completely recovered and was feeding normally by the following day.

Summary

The oral or parenteral administration of large quantities of sodium copper chlorophyllin to various experimental animals was completely without toxic effect. The only detectable effects in the intact animal were coloration of the fæces and, in the case of laying birds, the production of green-yolked eggs.

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TABLE I

The oral administration of sodium copper chlorophyllin

Species	Sex	Initial age	No. of animals	Daily dose (mg./kg.)	Duration (days)
Mouse	M/F	3 mths	6	2,500	7
Rat	M/F	21 days	30	2,000	123
Guinea pig	F	5 mths	2	1,000	7
Rabbit	F	4 mths	2	1,000	7
Dog	M	12 mths	1	1,000	7
Cat	F	2 yrs	2	1,000	7
Fowl	M/F	1 day	60	70	42
Fowl	F	1 year	8	500	17

TABLE II

Parenteral dosage with 2 per cent. w/v solution of sodium copper chlorophyllin

Species	Sex	Age	No. of animals	Route*	Dose (mg.) of chlorophyllin per kg. bodyweight
Mouse	M/F	3 mths	12	i/v	400
Mouse	M/F	do	6	s/c	500
Mouse	M/F	do	6	i/m	250
Mouse	M/F	do	6	i/p	1,000
Rat	M/F	do	2	i/v	250
Rat	M/F	do	6	s/c	500
Rat	M/F	do	6	i/m	250
Rat	M/F	do	6	i/p	1,000
Guineapig	F	5 mths	2	s/c	500
Guineapig	F	do	2	i/p	500
Rabbit	F	4 mths	2	i/v	200
Rabbit	F	do	2	s/c	60
Rabbit	F	do	2	i/m	60
Rabbit	F	do	2	i/p	500
Cat	F	2 years	2	i/p	60
Dog	M	1 year	2	i/v	200
Dog	M	do	2	s/c	200
Dog	M	do	2	i/m	50
Dog	M	do	2	i/p	200
Pig	F	3 mths	2	i/v	10
Pig	F	do	2	s/c	20
Pig	F	do	2	i/m	20
Pig	F	do	2	i/p	50
Fowl	M	6 wks	3	i/v	80
Fowl	M	do	3	s/c	40
Fowl	M	do	3	i/m	40
Fowl	M	do	3	i/p	50

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ARTERIOSCLEROSIS OF THE AORTA AND PULMONARY ARTERY OF AN OX DUE TO *CYSTICERCUS BOVIS*

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Introduction

ADVANCED atheroma-like lesions of the aorta and pulmonary artery of an ox believed to be caused by *Cysticercus bovis* have been observed. Advanced arteriosclerosis is a rare pathological lesion in domestic animals (Runnels, 1946). According to Creech (1941) only about 60 cases had been reported to the laboratories of the Bureau of Animal Industry in America during the preceding 25 years. The morbid anatomical and histological appearance of cysticercus infection in many organs have been described (Bacaloglu, Balan, Ballif and Vasilescu, 1929; Hoogland, 1930; Monnig, 1941) but the aorta and large vessels appear to have escaped attention.

Material and Methods

An ox heart containing numerous cysts, presumably parasitic, was sent by the Superintendent of the Slaughterhouse at Colombo for identification of the causative parasite.

A tentative diagnosis of *Cysticercus bovis* was made following examination of crushed preparations. The diagnosis was confirmed by the study of serial sections from Bouin and Formol-saline fixed materials.

The heart was preserved in Formol-saline for further investigation.

Serial sections of the aorta and pulmonary artery were made and stained with Delafield's Hæmatoxylin and Eosin, Van Gieson's stain and Weigert's Hæmatoxylin. Some sections were stained with Chlorazol Black E for elastic fibres and Crystal Violet for amyloid. Frozen sections were stained with Sudan III for fat.

Description

Macroscopic appearance.

The specimen consisted of heart, pericardium and a few centimetres of the aorta and the pulmonary artery. The pericardium was adherent in a few places to the epicardium and patchy thickenings and opacities were noted. Protruding from the surface of the heart and lifting up the epicardium were numerous translucent yellowish white dome-shaped cysts varying in diameter

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from 1 mm. to 10 mm. (Fig. 1). They were most numerous on the anterior surface of the left ventricle. On cutting into the heart the cysts were seen to be embedded in the myocardium and projecting on to the epicardial and endocardial surfaces. Within the heart the cysts were most numerous on the interventricular septum (Fig. 2).

Each cyst had a translucent white wall from a part of which an unarmed scolex projected internally, and contained a transparent straw-coloured fluid.

The aorta was rigid and inelastic. Its intimal surface was grossly irregular (Fig. 3).) Elevated button-like areas, pearly white or opaque yellowish white, alternated with depressed irregular scars and longitudinal wrinklings. Some of the elevations were stony hard. In others the centres were depressed. Calcified areas could be felt in the vessel wall.

The pulmonary artery showed similar changes.

Microscopic appearance.

The intimal outline was irregular due to the thickenings and depressed scars. The thickenings were due to proliferation of sub-endothelial connective tissue. The depressed scars were seen to be continuous with scar tissue in the media (Fig. 4). In the sub-endothelial tissue fibroblasts and histiocytes were present in large numbers. Cholesterol and lipid substances usually encountered in atheroma were not found.

The most striking changes were seen in the media. The muscle layers and elastic tissue fibres were interrupted and distorted by three types of lesions which appeared to be stages in the same pathological process. The first consisted of a blurring of outlines of muscle fibres, gradually fading of nuclei and replacement by a purplish mucoid-like substance. The second is the spindle-shaped collection of chronic inflammatory cells arranged between muscle fibres (Fig. 5). Individual cells consisted of fibroblasts, histiocytes, plasma cells and a few eosinophils. The third and the most striking lesion was the calcareous deposit (Fig. 6). Each of these areas of calcification conformed more or less to a definite pattern. A central cavity contained calcified material in the form of thin sheets, sometimes folded and crumpled, at other times cracked and broken up into smaller plates. In the smaller areas a ring of dark blue condensation was seen around the calcified plate. The cavity was surrounded by a capsule of laminated collagenous fibres and elongated fibroblasts. Further out a follicular arrangement of chronic granulation tissue, giant cells and eosinophils was seen. One such area of calcification is seen to connect by a tract of granulation tissue with a depressed intimal scar on one hand and a deeper area of calcification on the other (Fig. 4). The appearance suggests the invasion of the media from the intimal surface by a foreign material which then underwent calcification. These three types of lesions scattered irregularly completely disorganise the regular pattern of muscle and elastic tissue in the media (Fig. 7).

The adventitia was thickened and rather cellular. Cuffs of chronic inflammatory cells surrounded the vasa vasorum. Among the cells, eosinophils were prominent.

PLATE I

Fig. 1.
Numerous cysts are seen protruding from the surface of the heart. X 1/2.

Fig. 2.
Cysts in the interventricular septum. X 3/4.

Fig. 3.
The aorta is laid open to show the intimal surface deformed by elevations and depressed scars. X 4.

(Article by Seneviratne and Kulasiri, page 387)

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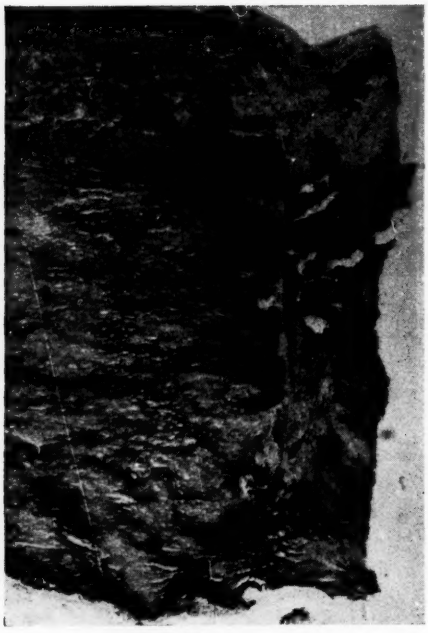

Fig. 4.
A low-power view of the whole thickness of the aorta. A depressed scar on the intimal surface connects with calcified areas deep in the intima by a tract of granulation tissue. H. & E. X 2.5.

Fig. 5.
A collection of chronic inflammatory cells lying between layers of muscle fibres. H. & E. X 108.

Fig. 6.
Calcified material surrounded by granulation tissue, giant cells, and eosinophils. H. & E. X 108.

Fig. 7.
The elastic tissue is interrupted and grossly disorganised. Chlorazol Black E. X 108.

(*Article by Seneviratne and Kulasiri, page* 387)

The pulmonary artery contained lesions almost identical in appearance macroscopically and microscopically.

The large veins did not show any such changes.

Discussion

Cysticercus bovis occurs chiefly in cattle but has been recorded in various other ruminants and even in man (Chandler, 1949; Monnig, 1941; Wardle and McLeod, 1952). Brumpt (1949) suspected a tropic response on the part of the larvæ to particular locations of the intermediate hosts. Wardle and McLeod (1952) suggest the possibility of some mathematical relationship between the diameter of the vascular and lymphatic capillaries determining such preferences. Whatever the reason be, blood vessels appear to be the less favoured sites. In the few references in the literature to lesions of blood vessels caused by *Cysticercus bovis,* no mention is made of the aorta or pulmonary artery. Hoogland (1930) reported hæmatogenous metastases of cysticerci in a pig. Particular attention was paid to alterations of the walls of blood vessels by the parasites. Bacaloglu, Balan, Ballif and Vasilescu (1929) described a hydatid infection in a young man where a metastatic dissemination of cuticular membranes from ruptured cysts in the heart took place. Nearly every organ in the body was involved—lesions in small and medium sized vessels are described. The basic lesion was calcification of emboli of cuticular membranes with giant cell reaction and encapsulation in chronic granulation tissue. There is no means of identifying with certainty calcified fragments of cuticular membranes. In the case reported here it was not possible to identify the calcified material but its appearance is suggestive of some structures such as the cuticular membrane. *Cysticercus bovis* cysts were numerous in the heart. It is reasonable, therefore, to suggest an invasion of the aorta and pulmonary artery by cysts or their contents.

Although macroscopic appearance bore a resemblance to atheroma, the microscopic picture was totally different. Maximal changes were in the media. Deposits of lipoid seen in atheroma were absent. The over-all picture was that of necrosis of the media with chronic inflammatory and giant cell reaction round calcified particulate foreign material.

A metastatic invasion via the vasa vasorum would result in maximal change in the adventitia and deeper layers of the media. But in the case under consideration maximal changes were in the intima and more superficial layers of the media. The alternative path of invasion would be directly through the intima. The presence of depressed scars in the intima leading down by tracts of granulation tissue into areas of calcified material suggests this path. Bacaloglu and others (1929) cite Deve as having demonstrated that hydatid scolices and brood capsules injected intravenously into rabbits were able to work their way out through the intima and media.

Fabris (1926*a*, 1926*b*) from experimental work was of the opinion that some undetermined substance diffusing out or excreted from *Cysticercus cellulosæ* was able to cause degeneration of muscle with which it came into contact. Similar

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substances may be present in *Cysticercus bovis* enabling it to work its way through the intima and media. The extensive destruction of muscle and elastic tissue appear to give further support to such a possibility.

The cysts within the aorta and pulmonary artery were degenerate whereas those embedded elsewhere had undergone normal development. This appearance may be due to one of two causes. Either the aorta and pulmonary artery by their anatomical structure prevent the development of cysts to maturity or the degenerate material is an embolism of ruptured cyst contents.

Summary

1. The aorta was rigid and inelastic, and its intimal surface grossly irregular with elevated stony, hard button-like areas alternating with depressed irregular scars.

2. The microscopic picture of the aorta was that of patchy necrosis of the media with chronic inflammatory and giant cell reaction round calcified particulate foreign material.

3. The pulmonary artery contained identical lesions.

4. The large veins did not show any such changes either macroscopically or microscopically.

5. The pathological picture suggests the invasion of the aorta and pulmonary artery by cysts of *Cysticercus bovis* or their contents.

6. The path of invasion suggested is through the intima.

7. The present study supports the view that cysticerci secrete or excrete substances capable of causing degeneration of muscles.

8. It is suggested that the structure of the aorta and large vessels prevent cysticerci from maturing.

Acknowledgments

We are grateful to Dr. D. W. Amerasinghe, Chief Veterinary Surgeon, Colombo Municipal Council, and Dr. S. Saravanamuttu, Superintendent, Slaughterhouse, Colombo, for the specimen; to Miss P. Jayawardena, of this Institute, and Mr. C. H. Fernando, of Christ Church, Oxford, for assistance with the literature; to Mr. R. Surendranathan for the photographs.

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A NOTE ON THE ANATOMY OF THE HORNS OF SHEEP

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It is only recently that any attention has been paid to the anatomy of the sheep, possibly because of its previous low economic value or more likely because of the anatomical similarity to the ox.

Chauveau (1891) mentioned that the horn of ox is nourished with blood supplied by two terminal branches of the palpebral artery. These two vessels run on the bone core of the frontal appendage where they are distributed almost exclusively to the generative membrane of the horny tissue. The palpebral artery comes off the medial branch of the superficial temporal which is in turn one of the two terminal branches of the external cartoid. Montane and Bourdelle (1917), Ellenberger and Baum (1932) and Sisson (1953) confirm these findings and all consider the sheep to have a similar supply. They also stated that the cornual branch of the lacrimal nerve supplied the horn.

Twenty sheep heads of different ages and sexes were dissected. The arteries and veins were injected with vinylite resin solution.

The dissections demonstrated:—

1. The os cornu or horn core. This is conical in form and is connected to the frontal bone through the frontal process. The frontal sinus extends to a variable extent into the bone core.

2. The vascular connective tissue layer. This covers the free surface of the bone core and separates it from the keratinised skin covering. It is continuous with that of the skin surrounding the horn.

3. The keratinised layer or horn covering. This is the outermost structure and consists of longitudinal fibres closely packed together.

4. The horn receives its blood supply from the following arteries:—

(a) The anterior auricular artery. This is the main blood supply to the horn. For the greater part of its course this artery is covered by the parotid gland, except behind the supraorbital process of the frontal bone, where it is covered by the auriculo-palpebral nerve and its branches. At the lateral side of the base of the horn, the anterior auricular artery terminates by dividing into two branches, the anterior and posterior cornual arteries.

At first the anterior cornual artery runs close to the horn base, then it crosses the ventral aspect of the anterior cornual vein where it divides into two terminal branches. One of these branches supplies the anterior and medial aspects of the horn, and has a branch running forward on the frontalis muscle which anastomoses with a similar branch from the other side of the head. The other terminal branch runs downwards on the frontalis muscle and anastomoses with one of the branches of the malar artery.

The posterior cornual artery runs round the posterior and medial aspects of the base of the horn. Its branches run upwards to supply the lateral, posterior and medial surfaces.

The branches of the anterior and posterior cornual arteries supply the bone tissue of the horn core passing through the foramina to anastomose. The horny

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covering is supplied with blood from the minute vessels from the anterior and posterior cornual arteries which penetrate the corium.

(b) The supraorbital artery (frontal artery). This artery originates from the ophthalmic artery before it enters the ethmoidal foramen. It then enters the supraorbital canal through its orbital opening dorsal to the frontal vein. On leaving the canal, the vessel divides immediately into several minute branches supplying the frontal and horn sinuses.

(c) The malar artery. The anterior auricular artery receives an anastomosing branch from the malar artery as it crosses the frontal muscle and therefore indirectly supplies the horn with blood.

5. The blood is drained from the horn by the following veins :—

(a) The anterior auricular vein is the main drainage of the horn. This vein is formed at the base of the horn by the union of the anterior and posterior cornual veins. At the level of the lower part of the base of the conchæ, the vein meets the transverse facial vein to form the superficial temporal vein which is one of the tributaries of the internal maxillary vein. It is covered, in its upper part, by the fronto-scutularis muscle and the auriculo-palpebral nerve runs along its dorsal surface. Throughout the rest of its course the vein is embedded in the parotid gland.

The anterior cornual vein is formed on the frontalis muscle and at the anterior part of the base of the horn the vessel receives several branches from the anterior and lateral surfaces of the horn. Immediately below the lateral part of the base of the horn, the anterior cornual vein meets the posterior cornual vein to form the anterior auricular vein.

The posterior cornual vein is formed behind the horn by the union of several small branches. It runs round between the ear and the posterior aspect of the horn, to meet the anterior cornual vein just below the lateral part of the base of the horn. In its course it receives branches coming from the medial, posterior and lateral aspects of the horn. Behind the two horns, tributaries of the right and left veins anastomose. Further anastomoses may occur between them and the facial and posterior auricular veins.

(b) The facial vein. This vessel is formed by several small branches on the frontal bone between the two horns. It runs downwards and forwards in an oblique direction and turns downwards around the external lacrimal fossa and continues on its course to join the external jugular vein. It receives in its course several branches coming from the horn. Some of the branches anastomose with those of the anterior cornual and posterior cornual veins.

(c) The supraorbital vein (frontal vein) is the main drainage of the frontal and horn sinuses. It is formed by very minute vessels joining together and passes through the supraorbital canal to join the ophthalmic vein in the orbital fossa. A fairly large branch passes through the supraorbital foramen to anastomose with the facial vein.

(d) The posterior auricular vein enters indirectly in the drainage of the horn by the anastomosing of some of its branches with those of the posterior cornual vein.

Fig. 1.

Nerves Supplying the Horn.

1, Facial nerve. 1′, Ventral buccal nerve. 2, Auriculo-palpebral nerve. 3, Dorsal buccal nerve. 4, Loop of the auriculo-palpebral around the anterior auricular vein. 5, Anterior auricular vein. 6, 12, 12′, 12″ and 13, Cornual nerve and its branches. 7, Loop of the anterior auricular nerve around the branch of the anterior auricular artery. 8, Anterior auricular artery. 9, Anterior auricular nerve. 10, Branches of the temporal nerve. 11, Anastomotic branch between the anterior auricular and cornual nerve.

Fig. 3.

Nerves, Arteries and Veins of the Horn.

1, Lacrimal nerve. 1′, 1″, 1‴ and 1⁗, Branches of the lacrimal nerve. 2, Frontal nerve. 3, Malar artery. 4, Anterior cornual artery. 5, Anterior cornual vein. 6, Facial vein. 7 and 8, Branches of the anterior cornual vein anastomosing with a branch of the facial vein.

Fig. 2.

Arteries and Veins of the Horn.

1, Superficial temporal artery. 1′, Anterior auricular artery. 2, Posterior cornual artery. 3, Anterior cornual artery. 4, Branch of the anterior cornual artery going to the lateral side of the bone core. 5, Transverse facial artery. 6, Malar artery. 7, Anterior auricular vein. 8, Posterior cornual vein. 9, Anterior cornual vein. 10, 10′, 10″ and 10‴, Radicles of the anterior cornual vein. 11, Transverse facial vein. 12, Facial vein.

(*Article by George, page* 391)

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[*Photo by Author*

[*Photo by Author*

The new headquarters buildings of the Fiji S.P.C.A., Suva

(*Article by Comben, page* 394)

6. The nerve supply.

(a) The auricular palpebral nerve originates from the facial nerve just below the base of the ear. On reaching the zygomatic arch, it forms a loop through which the anterior auricular vein passes and then divides into the temporal and the anterior auricular nerves.

The anterior auricular nerve runs upwards between the horn and upper eyelid covered by the frontalis muscle where it supplies several branches to the lateral and anterior parts of the base of the horn.

The anterior auricular nerve continues its course downwards on the frontal bone and may, above the upper eyelid, anastomose with the frontal and lacrimal nerve. Moreover, in its course above the zygomatic arch a branch anastomoses with one of the branches of the cornual nerve.

(b) The cornual nerve. This is a branch of the ophthalmic nerve which in turn is one of the three divisions of the trigeminal nerve. The cornual nerve reaches the lateral side of the base of the horn by passing below the posterior border of the supraorbital process and divides into two or three branches. One of these branches anastomoses with a branch of the anterior auricular nerve, while the remaining ones continue their course to the lateral and posterior parts of the base of the horn.

(c) The lacrimal nerve. This is another fairly large branch of the ophthalmic nerve. It divides into three or four branches which leave the orbital cavity by turning round the orbital edge of the frontal bone to gain its dorsal surface. The branches run upwards embedded in the frontalis muscle to innervate the horn supplying this muscle and the upper eyelid.

(d) The frontal nerve. This originates from the ophthalmic nerve close to the lacrimal nerve and sometimes the two nerves as well as the cornual nerve originate from the parent nerve by one stem. Its course resembles that of the lacrimal nerve and it also sends twigs to innervate the horn.

(e) There are three other nerves which could assist in innervating the horn indirectly. These are the first and second cervical nerves and the posterior auricular nerve. These nerves may anastomose with branches of the cornual nerve behind the horn base and thus indirectly take part in the innervation of the horn.

The above findings in the sheep illustrate how the arteries, veins and nerves differ in their course and distribution from the ox.

Acknowledgments

I wish to express my thanks to Professor T. Grahame, in whose department this work was carried out, for his advice and encouragement. My thanks are also due to the artist, Mr. E. D. Roberts, and to Mr. R. Hood for the photographs.

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ANIMAL WELFARE IN FIJI

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THE South Pacific island colony of Fiji consists of well over 300 islands, of which about 100 are inhabited; the two largest are Viti Levu and Vanua Levu, which between them carry 90 per cent of the total population of the colony. The capital, Suva, lies in a bay on the south-eastern corner of Viti Levu.

The native Fijians are of Melanesian descent and are famous for their fuzzy hair. They are also remembered by all who have visited the colony for their friendliness, and such is the welcome which they extend to all comers that they are now slowly but surely being outnumbered by the Indian population of the colony. The Fijians and Indians between them account for about 93 per cent of the total population, the remainder being Europeans and part-Europeans, and a small but significant colony of Chinese.

The indigenous wild animals of Fiji appear to be limited to a small grey rat and a " flying fox " which is in reality a bat. Dogs and pigs were introduced at an early stage, probably from Polynesia, and both of these animals now live in a wild state on many of the islands. Cattle, horses and sheep were introduced about the middle of the last century by settlers and missionaries.

There are attractive legends concerning the origin of the native names for these imported species. On seeing a pair of cattle landed the local natives are reputed to have asked what they were and, being told " a bull and a cow," to have coined the word " bulumakau " which is Fijian for cattle to-day. Similarly, the word for dog, " koli," is supposed to have come from " collie," and the word for pig, " pauka," from " porker," but, unhappily, it is probable that there is little truth behind any of these legends.

Sheep were introduced into Fiji during the 1850's and thrived for about fifteen years until outbreaks of disease and the cumulative effects of the hot, damp climate made further attempts at sheep farming uneconomic; there are now very few sheep in the colony. Zebu cattle and goats have been found to be better suited to the climate, and both have enjoyed a greater measure of success.

The mongoose was introduced from India during the 1880's to deal with wild rats which plundered the cane fields, but was quick to turn its attention to easier prey, attacking hen-roosts, native birds, and even shellfish; these animals are now a pest on many of the islands.

There are several varieties of snakes and lizards but all are harmless. The giant toad was introduced from Hawaii in 1936 to help to control slugs, beetles and millipedes, and this it does most effectively. There are no crocodiles in Fiji.

* * *

Perhaps the last place one might expect to find an active society for the prevention of cruelty to animals would be on a semi-tropical island group populated by a native race who are renowned for their kind and gentle nature and who keep comparatively few domestic animals. Yet in Fiji such a society

exists; the suggestion of its formation was first put forward by Mrs. D. Kirk shortly after the end of the last war, and the Fiji S.P.C.A. has been growing in stature ever since.

During its early years the Society was dependent on both the financial and moral support of the Government for its continued existence, and at the request of the Government it played a large part in the control of the island's populations of wild dogs by a policy of catching and destroying humanely as many of these animals as possible. In the latter part of 1953 the Society was able to report that most towns in Viti Levu were free from the nuisance of wild dogs, and that reports of stock having been destroyed by wild dogs were few and far between. A further onslaught was made in May, 1954, after which the Society reported:

> "A blitzkrieg was carried out, with three vans taking part, in the districts of Tavua, Ba and Lautoka. The patrol lasted for 14 days. During that month the Society's 'bag' reached the record figure of 2,017 dogs destroyed, bringing the total number of dogs destroyed to 8,337. Of these, 5,583 were females."

The Society was criticised by many of its would-be supporters for the part it was taking in this work. The policy of destruction was questioned by many "animal lovers" on moral grounds, despite the ever-increasing numbers of wild dogs and their menace to public health. In the latter half of 1954, however, the Society received timely notice from the Government that its assistance in this direction was no longer required. Simultaneously it received a number of congratulatory telegrams and its membership increased suddenly. The Society was then able to devote the greater part of its time and its funds to those more conventional activities which enlist the sympathy and support of the public. But the menace of the wild dog in Fiji has begun, once again, to increase alarmingly.

During these years the destiny of the Fiji S.P.C.A. was being shaped by another event: a supporter, Mr. W. W. Wright, bequeathed to the Society the little island of Nukudrau in Natewa Bay, which forms the eastern end of Vanua Levu. This island was sold in 1953 for £5,150 and the money was used to build a fine new clinic and headquarters building in Suva. To-day the Fiji S.P.C.A. boasts of premises which would put most of our animal welfare societies in Great Britain to shame, and the memory of the man who made them possible has been perpetuated by a simple plaque on the first of the range of dog kennels which reads:

> "NUKUDRAU.—In memory of W. Wright, who made this clinic and kennels possible."

* * *

The new headquarters of the Society in Walu Bay, Suva, were opened on January 26, 1955, by Lady Garvey, wife of the Governor of the colony. They consist of a waiting room, a consulting and operating room and pharmacy, and a large modern office. There are hospital kennels for 16 dogs and a cattery for 12 cats. Residential accommodation is provided for the secretary and

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manager, Colonel R. Walsh, M.B.E., M.M., B.Sc., and for a trained Fijian assistant. The clinic is visited daily by a veterinary surgeon who is paid a retaining fee by the Society; Mr. B. R. Cook, B.V.Sc., a graduate of Sydney University, is the first to hold this post.

In addition to its headquarters, the Society has organised six active branches; these are in the (mainly coastal) towns of Lautoka, Tavua, Ba, Nadroga, Nadi and Labasa. These are served by three modern hospital vans.

The work of the Fiji S.P.C.A. falls into the same two categories as that of all animal welfare societies: the provision of treatment for the animals of the poor, and the investigation of cases of cruelty with education or punishment of offenders.

The veterinary work carried out in the Society's clinics is mainly concerned with the domestic small animals and is similar to that of small-animal clinics all over the world: there are, however, some interesting differences.

Dog breeders consider Fiji a paradise when they learn that none of the dog virus diseases—including distemper and hard-pad disease—exist on the islands. Cats are not so fortunate as both virus influenza and gastro-enteritis occur frequently. But whereas dogs are fortunate with regard to virus diseases, almost every dog over the age of twelve months in Fiji is infested with heartworms; these filariæ reduce the average life of dogs in the colony to about six years. Heartworms are transmitted by mosquitoes, and the Society is co-operating with research centres in the U.S.A. who are investigating the development of the microfilariæ within the mosquito and also the identity of the species which are the responsible carriers. It has been noticed that cocker spaniels have a greater resistance to heartworms than other breeds, but it is not known whether this results from a special immunity or whether the dogs' long, thick coats simply render them less liable to be attacked by mosquitoes. Treatment of affected animals with antimony compounds is usually successful if started sufficiently early, but re-infestation occurs as soon as treatment is stopped.

Toad poisoning affects both dogs and cats in Fiji. Certain species of toads exude a poisonous toxin from their skins which, if licked by an animal, causes salivation, vomiting and convulsions. About 75 per cent of cases recover without treatment, however, and 100 per cent recoveries can be obtained if severe cases are treated with atropine.

Leptospiral infections of dogs are common, but jaundice never results. Ticks infest both dogs and cats, but there are no tick-born diseases. Another condition which commonly affects both of these animals on the islands is thought by the Society to be predominately an a-vitaminosis B_1; although alert, affected animals are said to suffer a generalised partial paralysis even to their vocal cords, so that their barks change in pitch. Such cases respond well to treatment with thiamin.

* * *

It is necessary only to see a list of some of the cases of cruelty which have been investigated by the Fiji S.P.C.A. during the last year to realise the necessity for this part of its work: a mare fell into a drain and was left to die during

nine days of thirst and starvation; 54 fowls were transported for three days at sea in a crate measuring only 30 in. x 54 in. x 30 in.; a horse was ridden with a bit made from a piece of barbed wire because "it had a hard mouth"; a bull was left for five days tethered by a piece of wire 10 feet long, which eventually pulled through its nostrils; native wharf labourers were found amusing themselves de-horning cattle on a ship by pulling their horns off with a loading net, while others were unloading goats by swinging them by their legs and throwing them over the ship's side into a railway truck; and so on.

Of the methods used by the natives for the slaughter of food animals, one correspondent has written:

"I witnessed a man belabour a pig for ten minutes with a heavy stick before the poor animal succumbed. On another occasion some natives were killing a cow. After hitting it several times on the head with an axe, it was on the ground half unconscious but still alive; one of the men then began to cut off one of its legs. With difficulty I persuaded him first to cut its throat."

The Society is now carrying out a thorough investigation into the methods of slaughter employed by the natives, but the practices which it finds most difficult to stop are those involved in the ritual preparation of various animals for slaughter before feasts, for their discontinuance would interfere with tribal customs: one such practice involves keeping a large turtle on its back for several days before a ceremonial feast.

It is also hoped that active steps may be taken in the near future to improve both the conditions in small local stock markets, and the conditions under which food animals are transported in overland vehicles and in inter-island vessels.

For a long time the Society was hampered in its efforts to control both individual cases of cruelty and general cruel mal-practices by existing legislation which required that active cruelty had to be witnessed before an offender could be punished. Passive cruelty—even the most flagrant cases of neglect cited above—could not be punished. In 1953, however, a new "Protection of Animals" Bill was introduced and passed by the Legislative Council; the shortcomings of the earlier legislation have now been amended, and a new era has dawned for the animals of Fiji.

* * *

The Fiji S.P.C.A. has an important job to do in the future and it is confident of that future. It looks forward to the day when it may be granted the privilege of Royal patronage and become, like its parent Society in England, another R.S.P.C.A.; when the signwriters were commissioned to paint the sides of the three new ambulance vans they were instructed to leave a space for an "R" in front of the "S.P.C.A." It looks forward, too, to the day when it may be financially independent and has a number of hardworking supporters striving in this direction—by no means the least important of these being a group of European ladies who run a bazaar stall in the town market every week-end.

The Society adopts an enlightened policy of animal welfare and prefers education to the punishment of offenders whenever possible. It does not believe that, except in a few isolated cases, either the Fijians or the Indians intentionally

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inflict cruelty on their animals, but rather that such cruelty is invariably the result of thoughtlessness or ignorance. The same problem arises in connection with the treatment of animals in the clinics. If the house dog or cat attached to a native family becomes ill it does not occur to them to take it anywhere to seek treatment. The majority of the patrons of the clinics are still Europeans. A few Indians may be seen at each session, but it is a rare event for a Fijian to be seen in the waiting room. The Society seeks to educate the native population to make use of the service which it offers, and an important part of this educational policy is a monthly magazine—" Kindness "—which enjoys a large circulation amongst children of all races over all the islands. It is estimated that it will take at least another three or four years before the Indians who are resident in the larger towns learn to make anything like full use of the Society's clinics, and probably more like ten years before the Fijians can be similarly educated. The success or failure of the Society will depend on the amount of confidence which its workers are able to impart during the next few years, while the native population are discovering what, to them, is an entirely new attitude towards animals.

ELECTROPHORETIC STUDIES OF ŒDEMA DISEASE IN SWINE

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THE electrophoretic analysis of blood sera from healthy animals is an effective method of demonstrating the concentrations of all the serum protein fractions present, within the physiological limits characteristic for a particular species. Disease may change this euproteinemia in varying degrees dependent upon the pathological changes taking place within the body. The pathologically changed serum protein pattern is called dysproteinemia. Euproteinemia and dysproteinemia have been extensively investigated in human medicine but few such investigations have been carried out in the field of veterinary medicine, no doubt because of the expense of the Tiselius type of apparatus. However, filter paper electrophoresis overcomes this difficulty as it is a simple and inexpensive technique. Boguth (1953, 1954) in his papers using the latter technique, presented normal and pathological serum protein patterns in domestic animals and stressed the diagnostic and prognostic value of this type of analysis.

This communication deals with both the protein picture in young, normal pigs and an attempt to show whether or not a characteristic dysproteinemia is associated with œdema disease of swine. Numerous reports have been published giving normal serum protein values for adult swine. A majority of the investigators used a free electrophoretic technique [Moore (1945), Deutsch, Goodloe (1945), Koenig, Hogness (1946), Svenson (1941)]. Boguth (1953), Kainrath,

Stockl (1945), and Hill, Schumann (1953) give values obtained by means of filter paper electrophoresis.

As œdema disease usually affects young pigs of between two and three months of age it is clear that the " normal " or " physiological " serum protein pattern of pigs of this age must be thoroughly established as a basis for comparison. There appears to be only one reference for the serum protein pattern in young pigs and this is a report by Jakobsen and Moustgaard (1950) based on a precipitation technique. Accordingly, to establish the normal protein values with the filter paper electrophoretic technique, a number of tests were run on apparently healthy pigs of this age group.

Material and Methods

The filter paper electrophoresis technique used in this work has been published previously by the author [Vesselinovitch, Funnell (1954)]. Diagrams were obtained by direct reading of stained strips on a photovolt densitometer. Relative values were calculated through planimetric measurements. No attention was paid to the so called " dye absorption factor."

" Normal " animals were clinically healthy pigs chosen at random from three different litters. There were in all eighteen such animals whose sera were tested at two and again at three months of age.

Sera of thirteen pigs suffering from œdema disease were obtained at intervals as they became available.

Results and Discussion

Jakobsen and Moustgaard (1950) have found a continuous change of serum protein patterns during the first six months of life. A/G ratio, which is 0.25 at birth, increases very rapidly in the course of the first month reaching a value of 1.5. Subsequently this decreases very slowly to about 0.40 at six months of age. γ— globulins, zero at birth, increase up to about 38 per cent during the first ten days of life, then decrease to 5 per cent by the thirtieth day, at which time the albumin value is 60 per cent and $\alpha + \beta$— globulins 35 per cent. By six months of age the albumin has decreased to 40 per cent, $\alpha + \beta$— globulins remain unchanged while γ— globulins have risen to 22 per cent. Boguth, using a filter paper electrophoresis technique, presented the following protein values in adult swine : Albumin 44.8 per cent, α— globulin 16.7 per cent, β— globulins 18.7 per cent and γ—globulins 19.8 per cent. It has been shown by Schoenheimer *et al.* [Schoenheimer (1942), Schoenheimer, Ratner, Rittenberg (1942), Shemin, Rittenberg (1944)] working with the isotopes C^{14}, N^{15} that the half life of protein molecules is about fourteen days. Thus one would expect that after three or four weeks there would be little γ— globulin left in the serum if the pig were unable to synthesize it. Thus, from the point of view of humoral defence, the above age would appear to be the critical, for during this period the γ— globulins, received via the colostrum, disappear very rapidly leaving only a small amount of autosynthesized proteins.

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Table I lists the values obtained from nine of the eighteen normal animals. The means and standard deviations for all eighteen animals at both two and three months of age are presented in Table II. Our results are in quite good agreement with those reported by Boguth (1953), except for the low γ— globulin fraction which can be attributed to the age of our pigs. However, these low γ— globulin values are as those given by Jakobsen and Moustgaard (1950). Thus

TABLE 1. SERUM - PROTEINS IN APPARENTLY HEALTHY ANIMALS

Number	∝A	∝0	∝1	∝2	∝3	β1	β2	γ1	γ2	A/G Ratio
2F1	44.8	2.4	2.4	12.9	-	12.3	13.5	7.0	4.7	0.81
2i	44.0	2.3	3.1	16.5	-	9.7	12.6	11.8		0.78
3A	45.7	2.3	3.5	12.7	-	11.9	13.4	5.6	4.9	0.84
2A	52.2	2.4	2.0	10.4	-	8.0	13.7	11.3		1.09
1A	43.7	2.2	2.4	13.1	6.9	6.2	13.8	6.9	4.8	0.77
B1	43.6	4.5	2.7	12.7	-	8.1	13.6	8.3	6.4	0.80
1i	45.7	2.1	1.6	16.4	-	8.5	12.9	6.4	6.4	0.84
ei	47.4	2.8	2.9	16.5	-	5.9	13.1	11.4		0.90
G1	44.5	1.5	1.5	19.5	-	10.4	13.2	9.4		0.80

TABLE 2. MEANS AND STANDARD DEVIATIONS OF SERUM PROTEINS IN YOUNG ANIMALS

	ALBUMIN	GLOBULINS			A/G Ratio
		∝	β	γ	
R E L %	45.85	20.44	22.42	11.29	0.86
±	2.6	2.4	2.4	1.6	

it was confirmed that the main characteristic of the "normal" serum protein pattern in pigs of the investigated age group is a low γ— globulin level.

The results obtained with sera from pigs sick with œdema disease are presented in Table III. For ease of presentation, the cases are divided into Groups I and II on the basis of the presence or absence of a clinically observable œdema.

These serum protein patterns show an increase of ∝— and γ— globulins in Group I but only of γ— globulins in Group II. With the globulin increase there was a concomitant albumin decrease which was especially marked in Group I (from 45.85 to 26.42). Accordingly the A/G ratio dropped down in Groups I and II from a normal of 0.86 to 0.35 and 0.59 respectively. The very marked decrease of albumin in Group I in itself suggests an explanation for the presence of œdema in this particular group.

Wuhrmann and Wunderly (1952) established the so called reaction patterns for serum proteins in human beings. Boguth (1952) presented the same patterns

in domestic animals. These patterns were arranged in different ways according to the increase of one or more of the globulin subfractions. We shall quote only the types that are of interest in this particular case. These are as follows:—

(1) Increase of α— globulin: Acute inflammatory process (particularly in dog and swine). (2) Increase of α— and γ— globulins with corresponding decrease of albumin: subacute or chronic inflammation (particularly in dog and swine). (3) Increase of γ— globulins: Chronic inflammation, cirrhosis of liver (dog), immunisation or R.E. System reaction.

TABLE 3. SERUM PROTEINS IN ŒDEMA DISEASE
(values in per cent of total protein)

	ALBUMIN	GLOBULINS							A/G Ratio
		α_0	α_1	α_2	β_1	β_2	γ_1	γ_2	
Group 1 (with Oedema)									
W	28.5	2.6	3.1	20.2	8.1	16.9	10.7	9.9	0.39
X	29.1	3.8	4.3	19.3	10.7	13.7	9.4	9.7	0.41
Y	28.5	4.5	4.5	19.3	13.7	12.5	10.2	6.8	0.39
I	31.2	2.5	1.2	21.2	8.7	13.7	21.5		0.45
II	25.2	4.8	5.7	21.1	9.6	11.5	22.1.		0.33
W-2	18.3	5.0	3.0	19.2	6.0	15.2	33.3		0.22
IV	24.2	4.3	4.3	21.4	10.0	13.0	22.8		0.31.
MEAN	26.42	27.89			23.32		22.34		0.35
Group 2 (without Oedema)									
A	39.7	3.1	2.7	15.9	7.3	13.2	6.9	11.2	0.65
B	37.2	2.0	2.1	19.3	5.8	8.7	24.9		0.59
C	36.6	2.2	2.3	16.2	7.6	13.1	22.0		0.57
D	38.0	2.7	1.9	11.1	8.4	14.2	23.7		0.61
E	36.0	2.7	2.2	20.1	6.5	11.6	20.9		0.56
F	35.3	6.1		19.5	6.1	12.2	20.8		0.54
MEAN	37.13	21.97			19.11		21.73		0.59

According to Selye (1950) a dysproteinemia, where albumin decreases and globulins increase (especially α— and β— globulins), is a symptom of the so called "adaptation syndrome." This dysproteinemia is thus a general manifestation of the organism to any kind of stress. Usually, this unspecific reaction is of a very temporary nature, being superseded by a specific type of dysproteinemia resulting from pathological changes in some organ, depending on the kind and intensity of stress.

A somewhat similar non specific dysproteinemia involving a reduction in albumin with a concomitant increase of globulins may, according to Wuhrmann (1952), result from the so called "tissue proteinuria" ("proteinurie in die

gewebe "). Such a tissue proteinuria is generally considered to be associated with damage to the blood vessels.

Conclusions

The small number of cases investigated makes it inadvisable to undertake a statistical analysis of the findings, and thus rules out the drawing of definite conclusions about the serum protein pattern in œdema disease of pigs. However, it can be stated that in the cases investigated there was consistent and definite decrease of albumin especially in Group I. This was consistently accompanied by an increase of α— and γ— globulins in Group I and only γ— globulins in Group II. The question as to what constitutes the immediate cause of the dysproteinemia remains unanswered. According to the serum protein patterns quoted above, Group I belongs to Boguth's type two, in other words these pigs might have been expected to have had a subacute or chronic inflammation. However, since microscopically there is no evidence of an inflammatory reaction in this disease [Schofield and Schroder (1954)] such an explanation cannot be accepted.

The lack of an alteration to the protein pattern of sufficient specificity to imply that the changes stemmed from a particular organ or organ system, makes it impossible to state the exact site and nature of the disturbances resulting in this dysproteinemia. However, it seems possible that the dysproteinemia in these cases is an instance of " tissue proteinuria "; being the manifestation of a non-specific reaction of the body to stress, accompanied by an enhanced activity of the reticulo-endothelial system.

Summary

(1) A low γ— globulin value is characteristic of the serum protein picture in young pigs. This confirms a previous paper reporting results obtained by the precipitation method.

(2) The sera tested from œdema disease sick animals show a decrease of the A/G ratio which results from a passive loss of albumin and an active increase of γ— globulins.

(3) A possible explanation for the dysproteinemia is presented.

Acknowledgments

The great help of Dr. F. W. Schofield (Department of Pathology, Ontario Veterinary College) is gratefully acknowledged, as it was through his considerable experience and active interest in " œdema disease " that all the samples of sera from affected pigs were obtained. The assistance of Dr. J. P. W. Gilman is also acknowledged for his constructive help in preparing this manuscript.

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AN EFFECTIVE TREATMENT FOR ANÆMIA OF PIGLETS

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YOUNG piglets may become anæmic in their first few weeks of life. This deficiency is due to two main factors, (a) the rapid growth of piglets with the concurrent increase in blood volume, and (b) the low amount of iron available from the sow's milk to supply a full complement of hæmoglobin to the increased number of red blood cells.

During the period of anæmia, which is most evident among stock bred, and those raised, in enclosed systems, resistance to infection becomes lowered and the piglets may suffer setbacks or die. Various treatments have been advocated to supplement the iron intake of the piglets. These supplements range from placing a sod of earth in the creep with or without soaking the earth with a solution of ferrous sulphate, dosing piglets with ferrous sulphate tablets, introducing reduced iron powder into the piglets' mouths, painting a mixture of molasses and ferrous sulphate on the sow's teats, to treating the pregnant sow with ferrous sulphate. The latter treatment does not increase the body reserves of the fœtus (Hart *et al.,* 1930). Most of the treatments, with variations, i.e., addition of copper salts or other supplements, have been found to have some effect but are messy and time consuming, and it is impossible to ascertain how much each patient ingests, absorbs and utilises.

London and Twigg (1952) working in these laboratories have succeeded in producing an iron-dextran complex designed for intramuscular use in the treatment of hypochromic anæmia in man. This paper reports our observations following a request to treat piglets by the same methods.

Materials used: (*a*) Iron-dextran solutions containing 3 per cent iron. Now increased in strength to 5 per cent iron and marketed as Imferon by Messrs. Benger's Ltd.; (*b*) a suspension containing 5 per cent iron phosphate.

Animals used: Suckling piglets of Essex Saddle-back or Large White x E.S.B. strains, commercially bred on an enclosed system.

Hæmoglobin estimations: These were made on blood samples taken from marginal veins of the ears and estimated by the oxyhæmoglobin method (King, 1951).

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Treatment and Results

Preliminary investigations showed the normal hæmoglobin values for adult pigs, on the farm where treatment was required, to be at approximately 12.0 g. per cent, and the levels for clinically affected piglets to be approximately 6.0 g. per cent. Using a formula, 1.3 x weight in lb.(15—Hb found), devised to calculate the amount of elemental iron needed for regeneration of Hb back to normal levels and to allow an extra 25 per cent on the normal Hb level for replacement of depleted body reserves, it was found that piglets would need 11.7 mg. iron per lb. of body weight. As we were using a 3 per cent iron solution we decided that a dose of 12.0 mg. iron per lb. would be a convenient one to use.

All our injections were made deep into the gluteal muscles of some 140 piglets. None of the animals showed any signs of discomfort.

TABLE I

Average haemoglobin levels for 8 untreated piglets

Age in days	Hb. g/100 ml.	Body weight lbs.	Total Hb. g. (B.vol.36ml/lb.)
10	6.9	6.5	16.4
19	5.68	7.25	14.9
25	5.37	8.2	15.8

TABLE II

Utilisation of injected iron dextran
(Average values for 8 piglets)

Time after trtmnt. - days.	Hb. g.per 100ml.	Body wt. lbs.	Total Hb (B.vol. 36ml/lb.)	Fe. injd. mg.	Gain in total Hb(g)	Mg of Fe required (1 g.Hb= 3.35 mg. Fe.)	Utilisation per cent.
0	5.1	11.75	21.6	140	-	-	-
0 + 7	9.6	14.0	48.3	-	+26.7	89	63.5
0 +14	9.8	17.25	60.8	-	+39.2	131	93.5

The results of our initial treatment of two litters were assessed entirely on the subjective improvement in condition of the piglets and were based on our own observations and those of the pig breeder. The piglets made a remarkably rapid change from a somewhat pathetic appearance to one of healthy liveliness, the improvement in condition being noticeable after only one week had elapsed following treatment. Subsequent litters were then employed to establish the optimum time for treating piglets. The effects of treatment were followed by observation of the hæmoglobin levels.

A proprietary food was made available in the creep for all piglets but it would appear from the figures presented in Table I that whatever small amounts of food, if any, were eaten during the critical anæmic period, very little or no

use was made of it towards hæmoglobin regeneration. We have assumed, therefore, that increases in hæmoglobin levels following our treatment of the piglets over this period could be attributed mainly to the iron administered intramuscularly, and Table II shows that almost the whole of the iron in the complex injected must have been absorbed completely and used for hæmoglobin regeneration within fourteen days of administration.

Fig. 1 demonstrates the average hæmoglobin levels of litters of piglets responding to injections on iron-dextran given on a weight/weight basis at different time intervals. Curve A shows that when piglets were dosed at three days old and consequently received a small amount of iron there was a response to treatment followed by a regression, whereupon a second larger dose of iron caused the hæmoglobin to rise to normal levels. Curve B shows a similar effect but with a larger initial response corresponding to the greater amount of iron

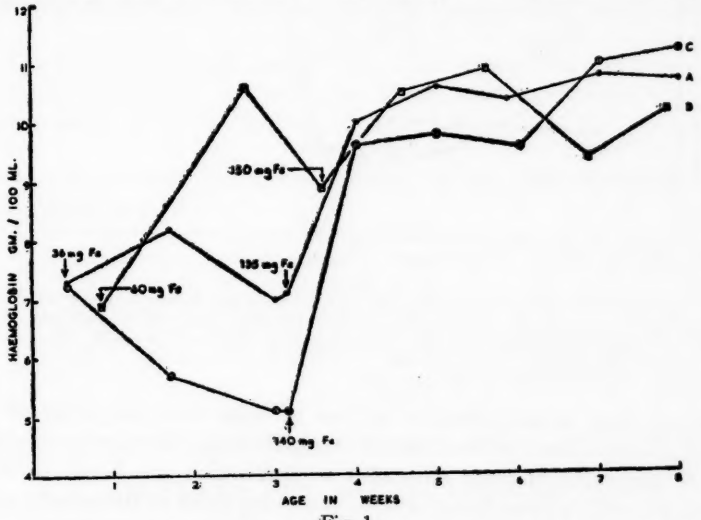

Fig. 1.
Average hæmoglobin levels for groups of piglets injected with 12 mg. iron per lb. bodyweight as iron-dextran at different ages.

given at six days old. When piglets were left untreated until three weeks old (Curve C) their hæmoglobin levels were quite low but a single injection at that time caused a marked initial response and subsequent return to normal limits.

During the course of treatment we gave equivalent doses of iron in the form of a suspension of iron phosphate to a few piglets. Three pairs of litter mates were treated (Fig. 2). One pair, "A," was given iron phosphate at eight days old. No response was seen within two weeks. Iron-dextran was then given and the hæmoglobin concentration attained normal levels within one week. The other two pairs were left untreated as controls until they were 25 days old when iron phosphate was administered to pair "C" and iron-dextran to pair "B." Both the "C" piglets died while with the pair "B" the fall in hæmoglobin concentration was halted and returned to normal within two weeks.

Post-mortem examination of the dead piglets of pair "C" showed that they had succumbed to a pulmonary infection and that at the site of injection there was a white deposit which on analysis proved to be iron phosphate in an amount equivalent to that injected. In contrast to the lack of absorption of iron phosphate, experimental work (Martin, *et al.*, 1955) has shown that approximately 90 per cent of iron-dextran is absorbed within 48 hours when given intramuscularly to rabbits.

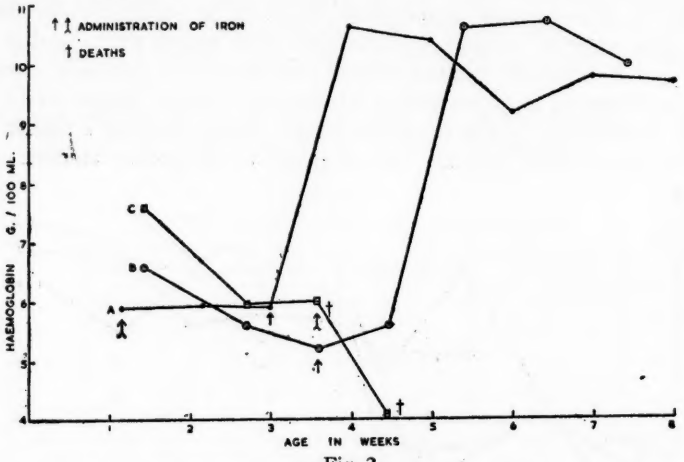

Fig. 2.
Average hæmoglobin levels of pairs of litter mate piglets injected with 12 mg. iron per lb. bodyweight as iron phosphate and/or iron-dextran.
A. Iron phosphate at 8 days old followed by iron-dextran at 21 days.
B. Iron-dextran at 25 days old.
C. Iron phosphate at 25 days old.

During these investigations a number of other iron complexes of polysaccharides were examined for their absorbability from the muscle site but the degree of absorption at seven days after administration was found to be only about 30 per cent, a dirty brown residue remaining fixed to the muscles around the injected site and showing a marked affinity for the muscle sheath. The residue was still present at 30 days after administration, whereas with iron-dextran there was only a faint yellow tinge at seven days, and at 30 days the muscles appeared normal.

Discussion

The loss of piglets due to secondary infections can cause considerable losses to pig breeders and a form of therapy which would help to keep piglets in a healthier condition should prove of value. We feel that we have shown that iron dextran could fulfil this requirement if it were available to the veterinary, as it is to the medical profession. It would appear that the anæmia is progressive up to at least five weeks of age and that piglets are likely to succumb to secondary infection during that time. It is desirable that treatment should commence early. If a dosage based on body weight is used very early in the neonatal period, then not sufficient elemental iron is provided to coincide with the rapid

growth and increased blood volume of the piglets. It is probably better to commence treatment shortly after the first week of life and give a second dose two weeks later. The preparations which we used contained three per cent iron but the material has now been improved and increased in strength to five per cent iron in order to keep the volume down to a minimum for injections. It is feasible that a single injection of 20 mg. iron per lb. could be given at about 10 days of age and thus provide a sufficiently high hæmoglobin level to tide the piglets over the critical period.

When using any type of parenteral therapy it is an advantage to know that the material will be absorbed and utilised completely. Some types of iron preparation which are absorbed only partially leave a marked dirty brown deposit distributed throughout the muscle to the detriment of the dressed carcase, even though the degree of absorption may be sufficient to cause some elevation of hæmoglobin level.

Summary

The treatment of piglet anæmia with a new preparation has been described. Iron-dextran is absorbed and utilised for hæmoglobin regeneration when injected intramuscularly to anæmic piglets.

Acknowledgment

We wish to thank the Directors of Benger's Ltd., Holmes Chapel, Cheshire, for permission to publish.

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THE EFFECT OF STILBŒSTROL IMPLANTATION IN COCKERELS ON BODY TEMPERATURE

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Literature

In poultry, as in all normal warm-blooded animals, the body temperature remains constant within relatively narrow limits. This homeothermy is attained because of an equilibrium between the heat produced by the animal, together with any heat it may absorb from its environment, and the heat which it emits. One of the main causes of a variation is the state of activity of the individual (Hutchinson, 1954). The body temperature rises after the ingestion of food, and is higher in animals maintained on a high plane of nutrition (Robinson and Lee, 1947). Body weight has an effect, for Fronda (1921) found that within a breed the heavier individuals tended to have a slightly lower temperature than the lighter, but sex appears to have little influence for he found no consistent differ-

ence between the body temperatures of cockerels and pullets at various ages from day old to 24 weeks.

Lorenz (1943) described the fattening action on cockerels of subcutaneously implanted diethylstilbœstrol pellets, and suggested its practical application. Since then this method has been widely adopted in this country and the United States as a means of improving the flesh of table poultry.

Experimental

During the years 1953-55, 175 Rhode Island Red x Light Sussex (RIR x LS) and 17 Buff Rock (BR) cockerels in 10 groups were taken at between nine and twelve weeks of age and weighed, and each group was divided into two equal pens of approximately the same weight. One pen in each group served as an untreated control, while each of the birds in the other pen had a 15 mg.

Table 1.

Details of the breed numbers, age at caponisation, live-weight at end of experiment, and average food consumption for each group.

Group No.	Breed	Numbers Capons	Numbers Cockerels	Age at caponisation (weeks)	Average live-weight at end of experiment (grms) Capons	Average live-weight at end of experiment (grms) Cockerels	Average food consumption per bird for the period (grms) Capons	Average food consumption per bird for the period (grms) Cockerels
1	RIRxLS	7	7	12	2250	2175	-	-
2	RIRxLS	5	4	12	2244	2215	-	-
3	RIRxLS	12	13	12	2434	2282	-	-
4	RIRxLS	11	13	10	2158	2073	-	-
5	RIRxLS	13	13	12	2450	2175	-	-
6	RIRxLS	12	11	10	1931	1931	-	-
7	RIRxLS	5	6	10	2215	2244	-	-
8	BR	8	8	9	2462	2259	7463	6350
9	RIRxLS	12	11	9	2432	2413	7142	7142
10	RIRxLS	10	11	9	2369	2336	7788	7444
Total		95	97					
Average					2314	2230	7464	6979

tablet of stilbœstrol dipropionate (Implantin, Burroughs Wellcome and Co. Brand) implanted in the neck with an Arnold injector. The caponised birds were watched for evidence of the regression or suppression of comb and wattle development which results from the suppression of the anterior pituitary gland by the œstrogen (Thayer, Japp and Penquite, 1945). This suppression or regression was quite obvious as it was accompanied by a paling of the comb, face and wattles. The capons and cockerels were housed separately in arks and runs which were moved on to fresh grass three times a week. A proprietary brand of growers pellets was fed *ad lib* at between 8.45 and 9.30 a.m. and 4.15 and 5 p.m. each day, and the quantities consumed by each pen were weighed for the last three groups. The birds were weighed weekly to see that normal growth was being maintained, and any bird which showed a rate of development markedly below the average was taken out of the experiment. Starting from the second week after caponisation, the temperatures of the birds were taken between 10.15 and 11 a.m. three days a week for six weeks. Stub-nosed half-minute clinical ther-

mometers marked in degrees Farenheit from 95 to 110 in units of 0.2 of a degree were used. They were guaranteed to be correct within 0.2° F., and were kept in position in the rectum for approximately one minute.

Discussion

In seven groups the temperatures were higher in the cocks than in the capons and in four of these the difference in the means was significant at the 1 per cent level, in two at the 5 per cent level, and in the other group it was not significant.

Table 2

Average body temperatures for each group over a six week period

Group No.	Average body temperatures °F Capons	Cockerels	Standard Error	Significance
1	106.6	107	0.156	Not
2	106.8	107.1	0.128	5%
3	107.4	107.3	0.073	Not
4	107.1	107.5	0.062	1%
5	107	106.9	0.196	Not
6	107.2	107.2	0.084	Not
7	107	107.3	0.008	1%
8	107.2	107.5	0.055	1%
9	106.9	107.1	0.048	5%
10	106.7	107.1	0.070	1%
Average	107	107.2	0.069	5%

Table 3

Average weekly variations in body temperature for all ten groups

Weeks after caponisation	Average body temperatures °F Capons	Cockerels	Standard Error	Significance
2	107	107.2	0.083	5%
3	107	107.2	0.089	5%
4	107.1	107.2	0.125	Not
5	107	107.2	0.111	5%
6	107	107.1	0.108	Not
7	106.9	107.2	0.088	5%
Average	107	107.2	0.069	5%

In one group the temperatures were the same, and in the other two groups the temperatures of the capons exceeded those of the cockerels but the difference in the means was not significant in any of these instances. The difference in the means of the average body temperatures of the capons and the cockerels in all the ten experiments was significant at the 5 per cent level of significance. (Table 2). The average weekly variations in body temperature for all the groups remained relatively level throughout the period (Table 3).

As the birds in both pens in each group were fed the same food and had

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their temperatures taken at the same time each morning, and as the times were approximately the same for each group, the food and feeding schedule would not have caused any variation. The average food consumption per pen showed that in one group the cockerels and capons consumed the same amount, but that in the other two the capons ate the most. The average body weights of the cockerels were lower than the capons in eight groups, the same in one, and higher in the other (Table 1). Neither of these differences was significant, but if either of these factors had influenced the body temperatures the evidence of the literature cited is that they would have tended to raise the temperatures of the capons rather than the cocks.

Although it could not be assessed, the capons appeared to be less active than the cockerels, and this lessening of physical activity was probably the factor responsible for the lower average body temperature for, as stated earlier, it has been established that the activity of an animal is one of the main causes of a variation in body temperature. It is probable, also, that this is one of the factors which contributes to the value of this method of caponisation in practice, for more of the food energy is utilised for fattening.

Summary

The temperatures of 95 Rhode Island Red x Light Sussex and Buff Rock capons and 97 cockerels kept under similar conditions in 10 groups were taken between 10.15 and 11 a.m. on three days in a week for six weeks. The results showed that the average temperatures of the cockerels were higher than those of the capons in seven of the groups, the same in one, and lower in the other two. The weekly average temperature for all 10 groups showed that the higher temperature of the cockerels, when compared with that of the capons, was significant at the 5 per cent level.

The writer wishes to thank Professor J. G. Wright for allowing the work to be undertaken, Dr. M. E. Castle for advice on the statistical interpretation of the records, and Miss J. Ashcroft for technical assistance.

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ABSTRACTS OF LITERATURE

THE USE OF A TRUSS IN PARAPLEGIA OF THE DOG, by R. J. Swarts and J. C. Jeffries, *Veterinary Medicine, L,* 319. July, 1955.

SWARTS and Jeffries provide a preliminary report of a useful truss for use in posterior paralysis of the dog due to trauma to the vertebral column. The truss is made of canvas, and has legbands passing under the rear legs made of elastic. It incorporates aluminium stays the length of the animal's body. It is suggested for use in disc injuries, some vertebral fractures, selected cases of vertebral dislocations and disorders of the sacro-iliac junction.

A Simplified Apparatus for Obtaining Semen from Dogs by Electrical Stimulation, by G. C. Christiansen and R. W. Dougherty, *Journal of the American Veterinary Medical Association,* 127, 50. July, 1955.

In view of the current interest in artificial insemination of the dog, it is of importance to note this description of a simplified apparatus for obtaining semen by electrical stimulation. The authors suggest that it will be particularly useful in cases where semen is required from stubborn males, which will not use an artificial vagina. The apparatus is simple, inexpensive, and will obtain good samples, without undue risk to the dog or the operator.

An Outbreak of Aujesky's Disease in Swine with Heavy Mortality in Piglets, Illness in Sows and Deaths *in Utero,* by W. A. M. Gordon and D. Luke, *Veterinary Record,* 67, 591. August 6, 1955.

Gordon and Luke of the Veterinary Research Department, Ministry of Agriculture, Belfast, provide a valuable and detailed account of an outbreak of Aujesky's Disease on a large commercial pig farm—maintaining approximately 500 sows. In this outbreak heavy losses were sustained in young piglets up to four weeks old whilst the virus also appeared to cause illness in nursing sows, as well as abortions or the production of mummified fœtuses. Also of interest is their finding that low vitamin A intake probably predisposed to the initiation of the virus infection and as a result of passage in such cases the virulence of the infection became enhanced. Rigid measures of isolation, and disinfection quickly eliminated the disease. This paper is particularly recommended for detailed study.

REVIEWS

Regional and Applied Anatomy of the Domestic Animal: Part I, Head and Neck, by J. T. Taylor. Edinburgh: Oliver and Boyd. Price 25s. net.

In this work, which will be completed in four volumes, the author has set out to describe the anatomy of the domestic animals as it concerns the clinician, concentrating only on those regions that are important in practice because they are the common site of injury or disease. If we may judge the whole from this first part, which deals exclusively with the head and neck, he has succeeded in producing something that will be of immense value at all stages of a veterinary career. In it the junior student, always sceptical of the utility of his preclinical studies, will find convincing demonstrations of the importance of anatomy in clinical work and these will not only stimulate his interest but also lighten his task. To the senior student and the practitioner this will be an invaluable source of reference to the applied anatomy of most of the conditions they meet and there will be few, no matter how experienced, who will not learn much of practical value from a study of these pages.

The layout is interesting and unusual in a British work. For the most part text and illustration are to be found on opposing pages, the figures being the

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central core of the work with the text arranged to explain and to amplify them. Some of the 104 illustrations are diagrammatic but most are drawn from dissections and are both accurate and at the same time emphasise only the salient features. The text is clear, concise and rarely elaborates beyond what is necessary to make its point.

Criticism will, of course, be made of the choice of subjects for illustrations, for naturally opinions will vary as to what is most important. On the whole, few will quarrel seriously with this selection. It may be that the dog receives too much, the farm animals too little attention: but then so far only the head and neck, on which elaborate surgery is practically confined to the dog, have been dealt with and doubtless the balance will be restored later. The reviewer would like to see more adequate treatment of the subarachnoid space and cerebrospinal circulation, the eye of the horse and the ligamentum nuchæ of the ox. These are small points. Veterinary students, teachers and practitioners can peruse this book with profit.

The appearance and production of the book are good and the spiral binding not too great an irritation.

THE ANATOMY OF THE SHEEP, by N. D. S. May. Brisbane: University of Queensland Press. Price £A2.

IN recent years the ruminants have occupied a more prominent place in the teaching of veterinary anatomy and the need for a reliable guide to their dissection has been widely felt. In so far as it satisfies this need the present work will be welcomed since it provides a concise and generally clear account of the anatomy, together with instructions for the dissection, of one of these species.

But in many ways this is a very disappointing work: in form it follows the traditional pattern, recording the anatomical facts impartially, paying little attention to what is, and what is not important, and carefully eschewing functional and clinical references. It is therefore difficult to read with attention and the rigid adherence to the formal terminology does not help to lighten the task. The illustrations form a vital part of any anatomical work: in this case they are not good. Photographs are rarely the most suitable medium for illustrating topographical anatomy and those reproduced are examples of this defect, for although generously sprinkled with labels it is difficult to distinguish the structures these identify. The remaining illustrations are line drawings and these are so reduced that they too convey little information.

The author promises a later edition in which the head and neck, at present omitted, will be described: perhaps it will be possible to enliven the second impression, for it seems a pity that so useful a work should be so dull.

The price, 40s. Australian, seems high, particularly in view of the rather poor production.

ANIMAL NUTRITION, by E. J. Sheehy, 1955. 732 p.p., with numerous illustrations. London: Macmillan and Co. Price 30s.

A REVIEWER criticising a new medical or scientific book has obligations both to the author of the book and to its potential readers. The author is entitled

to a fair appraisal of the results of his labours, and to have ambiguities, obscurities or errors pointed out, so that they may be rectified in later editions: the potential readers, as colleagues or students relying upon a reviewer's specialised knowledge, also have a right to a fair estimate of the book's value to them.

This large volume has both excellent and bad features. In a good but brief introductory chapter, Professor Sheehy does not reveal for whom it was written, whereas Dr. Hammond's foreword says that it is " primarily for Irish farming conditions," and the publisher's notice claims its use for veterinary students. From the frequent textual references to Irish conditions and practices, one must agree with Dr. Hammond, but, of course, good husbandry is international in its applications.

An almost complete absence of references to original literature suggests a book designed for undergraduate or technical student use. That apart, however, it is very difficult to know who will find the style of the book consistently easy. The farmer for whom Chapter 3 (" The Body Machine ") is suitable, is likely to be out of his depth with references to the " vaso-motor nervous centre," while technically equipped readers, e.g., veterinary students, may be irritated by references to " the body machine " and " food tube." The reviewer receives the impression that the author was rather undecided about his readers, with the result that the volume provides not one but several books. Thus, while much of the text is simple and lucid, there are sentences to be found such as . . . " There is some evidence, however, that an injection of it (vitamin C) improves the fertility of bulls in certain circumstances and possibly the same may obtain in the case of other farm animals in related circumstances." It is difficult to imagine who will benefit from reading such a sentence.

From the student point of view some repetition is good, but here again, a great deal of repetition suggests uncertainty of purpose. There are, for example, no less than nine references (pp. 133, 324, 464, 541, 557, 574, 627, 672 and 714) throughout the text to the relationship between sunlight, windows and vitamin D, three of them concerning pigs alone. One can understand a description of the origin of vitamin D appearing on page 20, but it is difficult to appreciate its appearance on page 714 also. In the latter case it occurs in a " supplementary chapter " of 24 pages entitled " Mineral and vitamin deficiencies and antibiotics ": apart from the four pages on antibiotics, the rest of the chapter is duplicated in earlier pages of Prof. Sheehy's volume.

The melting point of fats is discussed on page 8 but not defined until page 97, whilst students may be puzzled by the description of simple and mixed glycerides (page 8) as well as by the references to the " more soluble lipoid type of fat " which is " a more readily available form of fat than is ordinary neutral fat " (page 96). Moreover, the reference to " certain unsaturated fatty acids of the linoleic, linolenic and arachidonic types " will not leave students any better informed, since the last three named are not defined elsewhere, nor indexed.

Among the minor errors which have been noted are: " empirical " instead of " molecular " formula for carotene (page 16), " Carbohydrates are a course (? source) of body fat " (page 146)—not a happy phrase by any means—and

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central core of the work with the text arranged to explain and to amplify them. Some of the 104 illustrations are diagrammatic but most are drawn from dissections and are both accurate and at the same time emphasise only the salient features. The text is clear, concise and rarely elaborates beyond what is necessary to make its point.

Criticism will, of course, be made of the choice of subjects for illustrations, for naturally opinions will vary as to what is most important. On the whole, few will quarrel seriously with this selection. It may be that the dog receives too much, the farm animals too little attention: but then so far only the head and neck, on which elaborate surgery is practically confined to the dog, have been dealt with and doubtless the balance will be restored later. The reviewer would like to see more adequate treatment of the subarachnoid space and cerebrospinal circulation, the eye of the horse and the ligamentum nuchæ of the ox. These are small points. Veterinary students, teachers and practitioners can peruse this book with profit.

The appearance and production of the book are good and the spiral binding not too great an irritation.

THE ANATOMY OF THE SHEEP, by N. D. S. May. Brisbane: University of Queensland Press. Price £A2.

IN recent years the ruminants have occupied a more prominent place in the teaching of veterinary anatomy and the need for a reliable guide to their dissection has been widely felt. In so far as it satisfies this need the present work will be welcomed since it provides a concise and generally clear account of the anatomy, together with instructions for the dissection, of one of these species.

But in many ways this is a very disappointing work: in form it follows the traditional pattern, recording the anatomical facts impartially, paying little attention to what is, and what is not important, and carefully eschewing functional and clinical references. It is therefore difficult to read with attention and the rigid adherence to the formal terminology does not help to lighten the task. The illustrations form a vital part of any anatomical work: in this case they are not good. Photographs are rarely the most suitable medium for illustrating topographical anatomy and those reproduced are examples of this defect, for although generously sprinkled with labels it is difficult to distinguish the structures these identify. The remaining illustrations are line drawings and these are so reduced that they too convey little information.

The author promises a later edition in which the head and neck, at present omitted, will be described: perhaps it will be possible to enliven the second impression, for it seems a pity that so useful a work should be so dull.

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"borogluconata" for "borogluconate" (page 437). In an otherwise lucid account of energy metabolism, the omission to say for what species and physiological state Kellner's starch equivalents were devised will mislead students: "animals" here is too vague a term. The farmer who tries to remedy hypomagnesæmia or "nervous excitability symptoms associated with and akin to grass tetany" may find that "an injection of a magnesium salt is effective" in an undesirable manner (page 245). Furthermore, a farmer who tries to mix 0.05 per cent of cobalt sulphate or of potassium iodide into his own mineral mixture needs to be warned of the difficulties of uniformly distributing such small proportions of active agents.

After this list of faults of execution, it is still possible to praise Professor Sheehy's intention, which was clearly to describe livestock "in the round," for which purposes he first discusses nutrients and their metabolism, then looks at the animal in relation to its environment, passes on to discuss foods and general features of farm feeding and then spends some 400 pages on cattle, horses, sheep, pigs and poultry. The feeding of dogs and cats is not described. An index of some 10 pages is still not good enough for so large a book, for random inspection fails to find "animal protein factor" (page 250), "yeast" (page 233), though "dried yeast" is listed, or "unsaturated fat" (page 8). "Antibiotic" is listed, as are "aureomycin" and "penicillin," which makes the book's last sentence, "Experiments conducted in Ireland have confirmed the efficacy of a *particular** antibiotic in the prevention in scour of calves," uselessly and annoyingly vague.

The paper, printing and binding are excellent. There are some dozen figures, 15 formal tables and 43 photographs, a few of which are too small or ill-defined to show what the author intended. There are many points described about animal nutrition in Ireland which will not easily be found elsewhere.

* Reviewer's italics.

PROGRESS IN THE PHYSIOLOGY OF FARM ANIMALS, Volume 2, Edited by J. Hammond, 1955. London: Butterworth Scientific Publications. Price 45s.

THIS volume contains seven articles reviewing recent progress in certain fields of animal physiology and selected for their relevance to the problems of animal production. The titles and authors of the contributions are "Liveweight Growth" by R. W. Pomeroy, "Conformation and Body Composition" by H. Palsson, "Wool Growth" by H. R. Marston, "The Behaviour of Grazing Animals" by D. E. Tribe, "Sexual Behaviour" by A. Walton, "The Mechanism of Movement" by C. W. Ottaway, and "Egg Production and Fertility" by R. M. Fraps.

The list of contributors is sufficient to vouch for the authority and integrity of the articles which in many cases have the additional importance of being the sole reviews of these subjects to have appeared: most are supported by extensive bibliographies.

When completed, "Progress in the Physiology of Farm Animals" will

comprise three volumes. Two have now appeared and certain defects in the general form of the work have become apparent. Each individual contribution is of merit, but read together they lack coherence. Some range over a wide field and these inevitably are superficial; others probe more deeply into a restricted topic and will appeal only to specialists: several important subjects are omitted, while elsewhere material is included of which the practical importance and relevance is obscure; finally, there is a certain amount of overlapping, though considering the independent nature of the essays it is surprising that there is not more. These faults would seem to indicate insufficient editorial direction and an indecision regarding the category of reader for whom this work is intended.

None the less, there is much collected here which otherwise could be obtained only with great difficulty and labour, and which should be of interest to veterinary surgeons as well as to agriculturists. Workers in many fields will frequently wish to refer to these articles.

LEHRBUCH DER ENTWICKLUNGSGESCHICHTE DER HAUSTIERE, 2 Aufl. C. Zietschmann and O. Krölling, 1955. Berlin: Paul Parey. DM. 88.

IN the thirty years that have passed since the first edition, this has remained the accepted authority on matters of veterinary embryology: despite the wealth of recent research that is incorporated in a flow of modern textbooks this work has held its place because it alone is based on the development of the domestic animals. Even so, the need for revision had become urgent in recent years and in the welcome given to this new edition there will be a measure of relief that old loyalties need not be further strained.

The work now appears under joint authorship, and although based on the original version of Zietschmann it is very extensively altered. The text has been largely recast and expanded to bring it into line with recent advances: this is particularly noticeable in those parts that deal with gametogenesis, sexual cycles and placentation: all sections, however, have shared in the revision. There are now 597 figures: most are clear and well chosen to illustrate the text, but it is disappointing that none is in colour since this would have increased the clarity of the more complicated figures.

Minor criticisms of fact and of emphasis could be made but these would be insignificant in comparison with the value of this achievement. There is one major criticism—there are no references: this omission is surprising since it is obvious that the book, although primarily intended as a student's text, will be widely used by embryologists, by teachers of veterinary anatomy and by specialists in other fields. The authors' obviously unrivalled knowledge of the literature would have made a bibliogaphy of immense value.

In production and appearance this book fully maintains the high standard of its publishers.

ITEMS OF VETERINARY INTEREST

THE Institute of Biology announces that a symposium on "Biological Aspects of the Transmission of Disease" will be held at the London School of Hygiene and Tropical Medicine from September 29 to October 1, 1955. There

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are a number of papers of veterinary interest including "Factors affecting the transmission of coccidia and the development of disease in fowls" by Dr. C. Horton-Smith, and "Bacteria of veterinary importance and their transmission by non-biological agencies" by Professor R. Lovell. Full particulars can be obtained from the General Secretary, Institute of Biology, Tavistock House South, London, W.C.1.

We are pleased to learn of the appointment of Mr. D. L. Hughes, F.R.C.V.S., as Professor of Veterinary Pathology, at the University of Liverpool. He succeeds Professor R. E. Glover, M.A., B.Sc., F.R.C.V.S., who was recently elected Principal of the Royal Veterinary College.

Lederle Laboratories Ltd. have recently released Varidase to the veterinary profession in this country. This is an enzyme or chemical ferment for application to wounds which, through neglect, have become covered with dead tissue and hardened pus, and do not benefit fully from antiseptics. Varidase dissolves this matter and prepares the wound for treatment.

The Animal Health Trust have formulated a new drug, Gamahtine, for the immunisation of dogs against distemper and virus hepatitis. This has been prepared in conjunction with Burroughs Wellcome & Co., and initial success has been reported by the Trust's Canine Research Station. Gamahtine is stated to be active against secondary infections associated with distemper.

ECHO FROM THE PAST

(*From the* Veterinary Journal, *September*, 1880)

FOOT-AND-MOUTH DISEASE IN CAMELS

(*Extract from article by R. Pringle, M.R.C.V.S.,* Army Veterinary Department, Afghanistan*)

Among camels this widespread disease seems to me to prevail to a much greater extent than is generally believed; in fact, previous to my service in Afghanistan, I was of opinion that it did not exist at all, having been so informed. However, having now seen many thousand camels, and had time to notice them in health and disease, I believe it to be by no means an uncommon complaint. The symptoms in the camel, as in the elephant, differ from those presented by the cow, in a slight degree due, no doubt, to the difference in the conformation of the parts affected in the different animals. So far as my experience has gone, the feet of the camel suffer more, and the mouth less, than most other animals.

* Subsequently Major-General Sir Robert Pringle, D.G., Army Veterinary Corps.—*Editor.*

NOTICE

We have been asked to state that the paper by K. M. Dyce, "Experimental Bronchography of the Dog," which appeared in August issue, was submitted to us on March 28, 1955.